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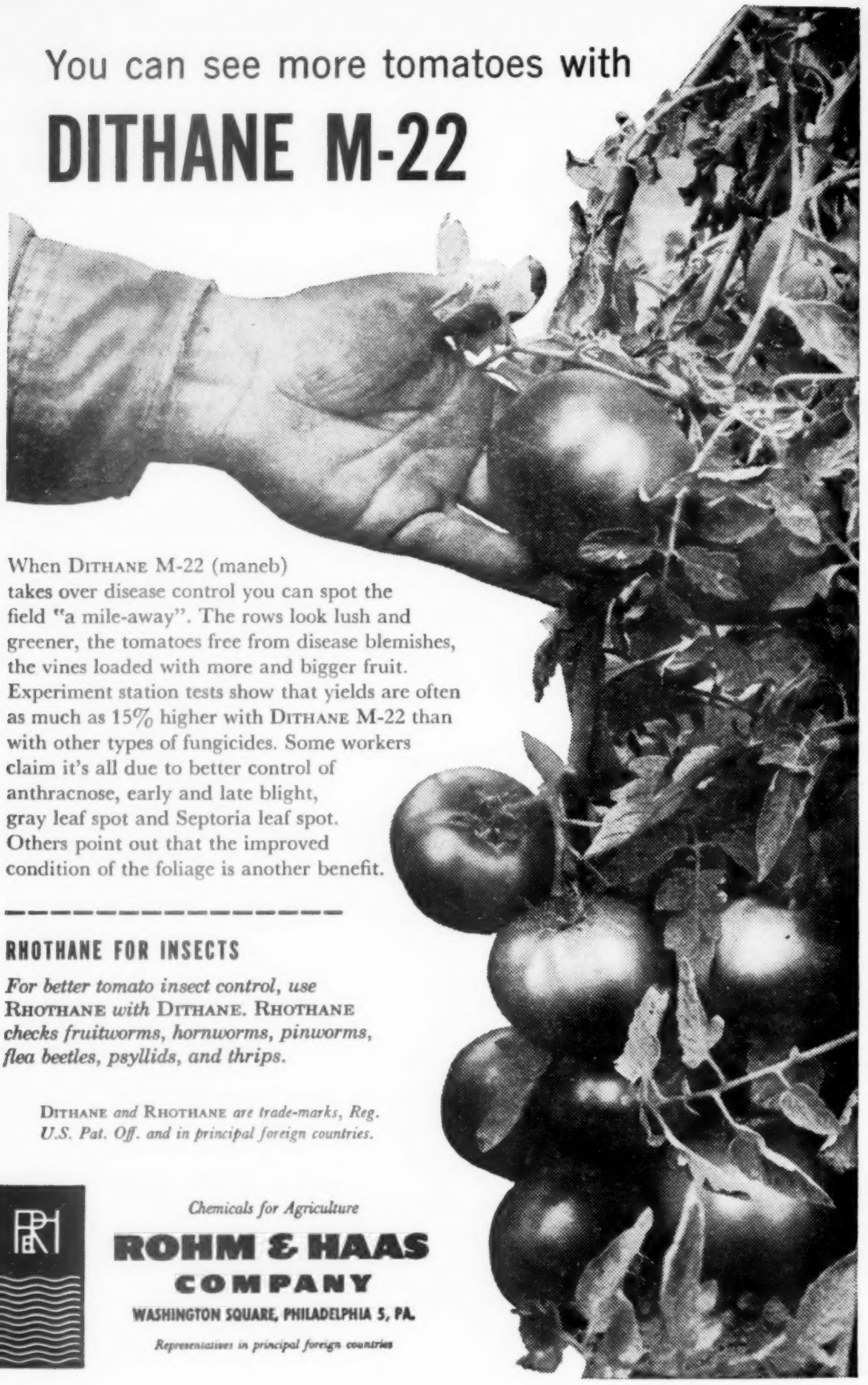

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AN IMPROVED METHOD OF DETECTING LOOSE-SMUT MYCELIUM IN WHOLE EMBRYOS OF WHEAT AND BARLEY[1]

W. Popp

SUMMARY

An improved method of preparing wheat and barley grain provides clear, undistorted whole embryos for rapid detection of hyphae of loose-smut mycelium: 1) kernels are boiled under pressure in 3% sodium hydroxide and 12% commercial water glass (sodium silicate) with 0.04% detergent to extract the embryos; 2) water glass is added to float off the embryos; 3) the embryos are boiled in 12% ethanol containing 15% sodium hydroxide; 4) heated in a 3:1 mixture of ethanol and glacial acetic acid to clarify tissue and remove oil droplets; 5) heated in 45% lactic acid; 6) heated in 45% acetic acid containing 0.1% trypan blue to stain fungus hyphae; 7) heated and mounted in 45% lactic acid. (Barley is dehulled by agitating in water in a modified homogenizer.)

Methods of detecting loose-smut infection in wheat and barley grain and determining the extent of invasion have had increasing application since development of the embryo-examination technique (3, 7). Information on invasion can be used to establish the acceptability of barley for seeding and to determine the need of control measures (2, 6). Data on the infection of wheat are of limited applicability because the embryos of certain varieties are susceptible to certain races of smut although the growing plants are resistant (3). Some of the weaknesses of existing methods (1, 2, 5) of detecting loose-smut infection in wheat and barley grain are remedied by the procedure described in this paper. The test should be especially valuable as a routine procedure in research and seed-testing laboratories.

DEHULLING.—Before extracting embryos of barley, the hulls surrounding the kernels should be removed for faster and more uniform penetration of the treating chemical. The only equipment required is an electric homogenizer (Waring Blendor) fitted with a power-control instrument (Powerstat). The cutter knives of the Blendor are sheathed in tight-fitting rubber tubing ($\frac{1}{8}$-in. bore, $\frac{1}{16}$-in. wall) after the blades are shortened about $\frac{3}{16}$ in. and the knife edges dulled.

In practice, up to 600 grains are placed in the Blendor jar with about 250 ml of distilled water. With the "Powerstat" the motor is gradually accelerated from rest to operation at 50 volts for 10 minutes. The contents of the jar are then rinsed in several changes of water, the rate of draining being so regulated that the kernels remain in the jar while the hulls are washed away.

EXTRACTION AND SEPARATION OF EMBRYOS.—Embryos should be extracted in a beaker or similar straight-sided container. Aluminum discolors the solutions, and should not be used. For 25 kernels a container basal area of about 17 cm^2 is large enough to permit easy visual inspection of individual kernels in completing the process. The processing solution should be at least 1 in. deep.

The solution consists of a mixture of 3% sodium hydroxide and 12% commercial water glass (sodium silicate) to which is added liquid soap or a detergent (about 0.04% by volume). The sodium hydroxide dissolves the starch grains and causes the embryo and endosperm to swell; the water glass prevents injury of the swelling embryo when it ruptures the pericarp; the soap or detergent prevents foaming and promotes fast and uniform penetration of the processing solution.

The grain and solution are heated at 5 lb. pressure in a pressure cooker—barley for 30 minutes and wheat for 60 minutes. The resulting liquor is stirred to free any embryos adhering to the viscous remains of the endosperm. Water glass is then added until the embryos float and can be skimmed off. Finally, the embryos are rinsed in water.

CLEARING.—Clearing decolorizes the tissues and removes oil droplets, leaving the embryos transparent. The clearing procedure is a modification of that described earlier (4).

The embryos are then heated 45 minutes at 5 lb. pressure in 12% ethanol containing 15% sodium hydroxide. After washing 15 minutes in several changes of water, the embryos are again heated at 5 lb. pressure, this time for 1 minute in a 3:1 mixture of ethanol and glacial acetic acid. Finally, they are heated at 5 lb. for 1 minute in 45% lactic acid.

STAINING.—The cell walls of fungus hyphae in the embryos are stained by heating 1 minute at 5 lb. pressure in 45% acetic acid containing 0.1% trypan blue. Excess trypan blue is removed by heating 1 minute at 5 lb. in 45% lactic acid, which is also used as the mounting medium.

MOUNTING.—The embryos are mounted on a rectangular slide ($\frac{3}{16}\times2\times3$ in.) made of transparent sheet-plastic (Lucite) having a surface grooved parallel to the long axis. The grooves are $\frac{1}{32}$ in. deep, $\frac{3}{32}$ in. wide, and $\frac{1}{16}$ in. apart. The embryos, poured on such a slide along with a little of the 45% lactic acid, tend to become arranged in parallel rows as they settle to the bottom of the grooves, and can be examined conveniently.

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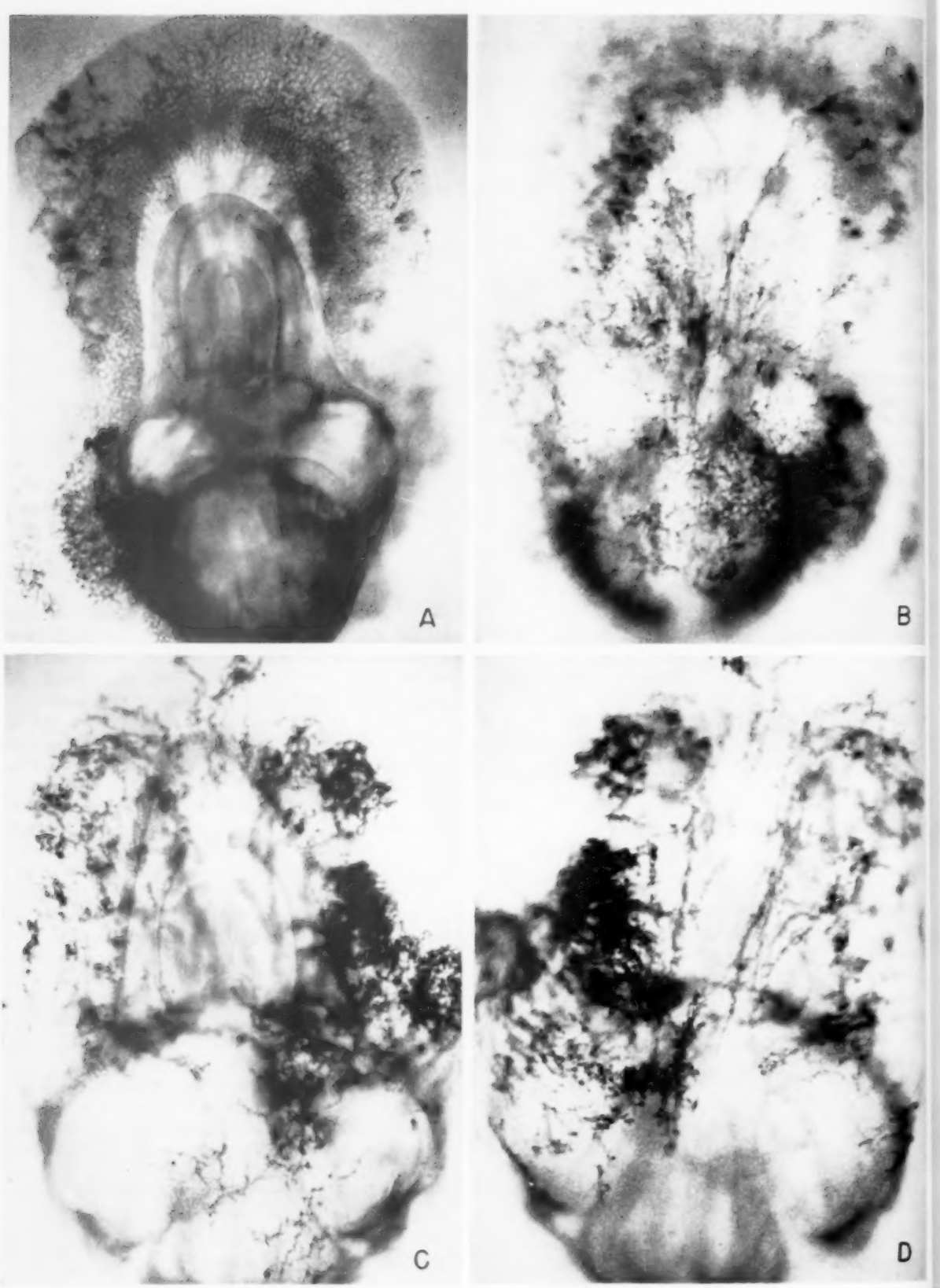
Contribution No. 2 from the Canada Department of Agriculture Research Laboratory, Winnipeg, Manitoba.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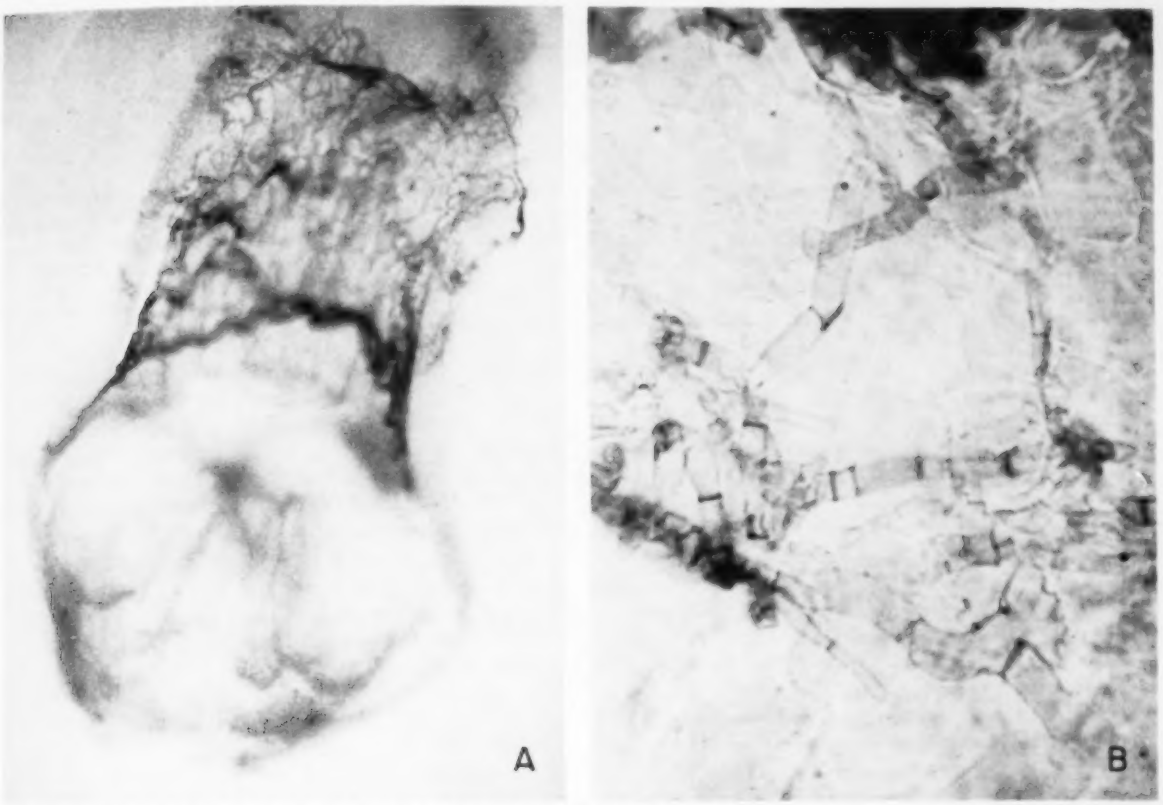

FIG. 2. A) Infection in a whole embryo of barley by fungus mycelium other than loose smut (×46). The hyphae of the fungus are concentrated in a stringy weft over a portion of the embryo. B) Detailed view of hyphae of unidentified fungus (×700).

EXAMINATION, DETECTION, AND DIAGNOSIS.—Microscopic examination at about 40-60× permits the widest possible field of view without loss of too much detail. Because of the clarity of the tissue, smut hyphae on the surface or within the embryos are detectable from any angle (Fig. 1). When an invasion does not appear typical of loose smut (Fig. 2A), higher magnification is advisable (Fig. 2B).

DISCUSSION.—The routine test here described was designed to permit an accurate and rapid detection of loose-smut infection in embryos of barley and wheat. The entire process, including dehulling (barley), extracting, separating, clearing, staining, mounting, and examining, requires but a few hours in a single day. Embryos are not damaged or lost in processing. One of the most important advantages is the resultant clarity of the tissue. This permits detection of smut mycelium without macerating or flattening the embryo. Estimation of the extent of invasion is therefore accurate and rapid.

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←

FIG. 1. Loose-smut-infected embryos of wheat and barley prepared by the improved embryo-examination technique and seen from different angles (×46). A) Face or axis, and B) scutellar views of a wheat embryo. Hyphae (not all in focus) of the smut fungus appear as darkly stained patches and irregular streaks in the transparent host tissue. C) Face or axis, and D) scutellar views of a barley embryo. Hyphae appear as darkly stained patches and streaks.

A STUDY OF THE PROGENY FROM THE SELF-FERTILIZATION OF RACE 111 OF PUCCINIA GRAMINIS VAR. TRITICI[1]

Roy D. Wilcoxson and K. D. Paharia[2]

SUMMARY

When race 111 of *P. graminis* var. *tritici* was selfed, 15 different races were identified among 23 isolates from the progeny, 8 of them appearing to be new races. Of the 7 resembling previously described races, only one resembled the parent culture. Lee, Khapli, and Vernal were resistant to all isolates; Little Club was susceptible to all. Marquis, Kota, and Reliance were susceptible to most isolates; the durum wheats and Einkorn were resistant to most.

Recent studies (1, 7, 8, 11) have shown that new races of the wheat stem rust fungus may arise from mixtures of urediospores of existing races. Race 111, used in some of those investigations, is particularly well-suited to this type of study because all of the standard stem rust differential varieties, except Little Club, are highly resistant to it. Nothing has been known about the genetics of pathogencity of race 111, nor of other races used in mixtures, but information on inheritance of urediospore color and pathogenicity (5, 6, 9) of *Puccinia graminis* Pers. var. *tritici* (Eriks. & E. Henn.) Shear, Ball, Jacks., & Stakman has been most helpful in designing experiments. An understanding of the inheritance of pathogenicity of race 111 might be expected to throw light on the question of heterocaryosis as a source of new races, and with this in mind, a study of progeny resulting from the self-fertilization of race 111 was undertaken.

MATERIALS AND METHODS.—Telia of race 111 obtained from Dr. I. A. Watson were used to furnish material for selfing this race on barberries. The methods used were essentially those described by Johnson (2, 4). When uredia from inoculations with aeciospores were obtained, masses of spores from single uredia were transferred to Little Club seedlings and, when the inoculum had sufficiently increased, the isolates were identified on the standard stem rust differential varieties (10) plus variety Lee. If infection appeared to be the result of a mixture of isolates, single uredia were again increased, and the test was repeated with the additional isolates. When a uniform reaction was produced on the differential varieties, the test was repeated 2–3 times before the reaction was considered typical of an isolate.

RESULTS.—When 23 single-uredium isolates were compared with parent race 111, 15 different races were identified (Table 1), 8 of them appearing to be completely new races. Seven of the races resembled previously described races of *P. graminis* var. *tritici* (10). Isolate F resembled the parent, but was not the same. Isolates A and H resembled, respectively, races 148 and 3, except that they did not produce a type-3 infection on Acme. Isolate G was like race 12, but did not produce a type-3 infection on Einkorn. Isolate N resembled race 153, but did not produce a type-X infection on Vernal. Isolates B and I were practically identical to races 187 and 54, respectively.

Lee, Khapli, and Vernal were resistant to all 15 races; Little Club was susceptible to all of them. Marquis was susceptible to 11 of the 15 races obtained, Kota to 12, and Reliance to 12. The durum wheats were resistant to most of the races: Arnautka was resistant to 9, Mindum to 13, Spelmar to 12, Kubanka to 14, and Acme to 11. Einkorn was resistant to 11 of the races.

DISCUSSION AND CONCLUSIONS.—Race 111 proved to possess genes that enable the fungus to incite susceptible infection types on all the differential wheats except Lee, Khapli, and Vernal. Even though the progeny populations were small, comparison of the data with those of Johnson (4) reveals still other unusual characteristics of race 111. Although self-fertilization of races of wheat stem rust usually yields progeny that resemble the parent on Marquis and Kota, such was not the case with the progeny of race 111. Further, resistant infection types incited by race 111 on Arnautka, Mindum, and Spelmar appeared to be dominant, which is most unusual for North American races of wheat stem rust. Only on Reliance and Einkorn did the progeny behave as if the parent were a race of *P. graminis* var. *tritici*. Then, factors enabling race 111 to incite resistant infection types appeared to be dominant.

The parent race 111 may have been a hybrid of *P. graminis* var. *tritici* with *P. graminis* Pers. var. *secalis* (Eriks. & E. Henn.) Seym., and the unusual behavior of parent and progeny may have been due to inhibitors, as suggested by Johnson (3). Thus, inhibitors suppressed pathogenicity of the parent to the various wheats, whereas inhibitors in the progeny were not effective on Marquis, Kota, and Reliance, but were generally effective on Acme, Arnautka, Einkorn, Mindum, and Spelmar.

Regardless of the mechanism, the results help explain the origin of Watson's (11) new races, NR 3 and 34, which incited susceptible infection types on the durum wheats although they had originated from

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TABLE 1.—*Reactions*[a] *of 13 varieties of wheat to 23 biotypes of the stem rust fungus resulting from self-fertilization of of race 111*

Variety	Race 111	Progeny from selfing race 111														
		A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
Little Club	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Marquis	0;	4	3	4	2	2	0	3	4	3	4	3	3	3	0	4
Kota	0;	4	3	4	3	4	0	3	3	0	X	X	X	X	0	3
Reliance	0;	4	4	1	3	4	0	4	4	0	4	4	3	3	4	4
Arnautka	0;	0	3	0	3	X	0;	3	0;	0	3	0	0	0;	0;	4
Mindum	0;	1	3	0	1	1	0	0	1	0	X	0;	0;	0;	0;	1
Spelmar	0;	0	1	1	1	3	0	0	1	0	3	3	0;	0;	0	0
Kubanka	0;	0;	0;	0	1	0;	0	0	0;	0	3	1	0	0;	0	0
Acme	0;	0;	0;	0	3	1	0	3	0	3	0;	3	0	0;	1	0;
Einkorn	0;	0	0;	0	0;	1	0	0	3	3	0;	3	0	3	0	0
Vernal	0;	1	0;	0	0;	0;	0	0;	0;	0	0;	0;	0;	0;	0;	0;
Khapli	0;	1	1	0	1	1	0	0;	1	0	1	1	1	1	1	1
Lee	0;	1	1	0	0;	1	0	0	0;	0	0;	0;	0	0;	1	0
No. of isolates		2	1	1	1	1	1	1	1	1	1	1	7	1	2	1

[a] Infection types are as follows: 0 = immune; 0; = flecks; 1 = very resistant; 2 = moderately resistant; 3 = moderately susceptible; 4 = very susceptible; X = heterogeneous (10).

urediospore mixtures of races 111 and NR 2, to which the durum wheats are highly resistant.

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EFFECT OF TEMPERATURE AND INOCULUM SOURCE ON THE MOVEMENT OF THE INTERNAL CORK VIRUS AND ON SYMPTOM DEVELOPMENT IN CORE-GRAFTED SWEETPOTATO ROOTS[1]

A. S. Williams and L. W. Nielsen

SUMMARY

Successful transmission of internal cork virus of sweetpotato into healthy roots from core-graft inoculations is influenced by temperature and the presence of necrotic tissue in the core. The virus moved 1 in. from the core-graft in 60 days at 60°F, 14 days at 70°F, and 6 days at 80°F. About 50% of inoculated roots developed symptoms in 46 days when the inoculum core contained necrotic tissue, but only about 15% when it did not.

Internal cork virus is often transmitted from one sweetpotato to another by core-graft inoculation as adopted by Nusbaum (8). Using this inoculation method (7), the junior author has sometimes obtained variable results. Harvested roots from some sprouts developed numerous, well-defined symptoms, whereas roots from other sprouts were free of symptoms. This response suggested that the virus had not yet become systemic in the inoculated root when the sprouts were removed. Generally, cores from infected roots are used as inoculum without regard for the presence or absence of necrotic tissue within the cores. Aycock and Hughes (2) obtained a lower rate of transmission with tissue from symptomless carriers than with tissue from Porto Rico sweetpotatoes containing necrotic lesions. However, they did not compare the two sources of inoculum from diseased Porto Rico roots.

The temperature at which core-grafted roots are incubated may also influence virus movement in the root. Several investigators (1, 4, 5, 10) have demonstrated that naturally infected roots stored several months at 70–75°F showed an increase in number and size of lesions, whereas similar roots at 55–60°F developed few symptoms beyond those observed at harvest. Roots grown in artificially heated soil have more lesions at harvest than roots grown under natural field conditions (6).

To gain information on the movement of the virus, core-graft-inoculated roots were incubated at different temperatures, and virus movement was bioassayed at intervals. The influence of inoculum source on virus movement and lesion development was studied by inoculating healthy roots with infected cores that did or did not contain necrotic tissue.

MATERIALS AND METHODS.—Roots of Porto Rico sweetpotato were used for all tests. Roots free of internal cork were produced on an isolated coastal island in Core Sound, off North Carolina. To check on freedom from internal cork, about 1/10 of each crop was examined for root symptoms after 5–6 months at 75°F. This stock of roots has remained free of internal cork for 7 years. Roots selected for graft inoculation were 6–8 in. long and weighed 6–10 oz. Cores of inoculum were cut from infected roots showing internal necrosis. In some tests, cores containing necrotic tissue were distinguished from cores without.

Grafting procedure consisted of removing cores of tissue with a number-5 cork borer and inserting them into holes cut with a number-4 cork borer. The cores were 1 in. long.

EXPERIMENTAL.—*Influence of temperature on virus movement.*—Inoculated roots were incubated at 3 temperatures, and virus movement bioassayed at various intervals. This test involved 2 core-graft inoculations. The first, herein called the "initial" inoculation, consisted of inserting cores of infected tissue into healthy roots. After roots receiving the initial inoculations had been incubated for various intervals, cores of tissue were cut from them and inserted into other healthy roots to assay internal cork virus away from the core graft. This second inoculation is referred to as the "bioassay" inoculation, and the roots as "bioassay" roots.

In the initial inoculation, healthy roots received, on the root end, cores of infected tissue not selected for presence or absence of necrotic tissue. The inoculated roots were equally divided and bedded in 3 flats of steamed, moist soil, and each flat was incubated at a different temperature: 60, 70, and 80°F. Virus movement from cores into inoculated roots was determined after the following incubation periods: 45–48, 60, and 70–75 days at 60°F; 14, 20, 27, and 34 days at 70°F; and 6, 13, and 21 days at 80°F.

For each bioassay, 10 inoculated roots were removed from the soil, and cores of tissue were cut at distances 1, 2, and 3 in. away from the initial inoculation. These cores were inserted individually into other virus-free roots. Bioassay roots were bedded in flats of steamed soil and incubated 4–5 months at 85–95°F in insect-free cages in a greenhouse. The soil was kept moist, and sprouts developed from the inoculated roots.

Two control inoculations were included to check on the bioassay inoculation. One consisted of inserting virus-free cores into healthy roots, and the other consisted of inserting cores of infected tissue into healthy roots.

Development of internal cork symptoms in bioassay roots was interpreted as evidence of virus movement to the distance from which the cores of tissue were

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TABLE 1.—*The effect of incubation temperature on movement of internal cork virus in core-graft inoculated sweetpotato roots*

Incubation °F	Incubation Days	Test site [a] (in.)	Infectivity [b] (%)
60	45 & 48 [c]	1	0
		2	0
		3	0
	60	1	18.7
		2	3.0
		3	9.4
	70 & 75	1	37.5
		2	20.8
		3	16.1
70	14 [d]	1	20.0
		2	0
		3	0
	20 [e]	1	37.0
		2	20.0
		3	4.3
	27	1	40.0
		2	14.3
		3	15.1
	34	1	24.1
		2	22.6
		3	12.1
80	6	1	2.6
		2	5.4
		3	0
	13	1	12.5
		2	15.6
		3	2.8
	21	1	16.7
		2	21.2
		3	3.2
Control inoculations:			
Healthy roots inoculated with healthy cores			0.0
Healthy roots inoculated with infected cores			54.7

[a] Distance from initial inoculation bioassay cores removed.
[b] Percentage of bioassay roots that developed symptoms.
[c] Roots assayed for virus movement in 2 tests.
[d] Roots assayed for virus movement in 1 test.
[e] Roots assayed for virus movement in 3 tests.

taken. Each root was sliced and carefully examined for internal necrosis. The data from 4 tests are summarized in Table 1. The data are based upon examining 30–40 bioassay roots, except where fewer than 4 tests were performed.

The development of internal cork symptoms in bioassay roots was markedly influenced by the incubation temperature of roots that received the initial inoculation. The influence of incubation temperature on virus movement is illustrated by time elapsed while the virus moved 1 in. from the initial graft: 60 days at 60°F, 14 days at 70°F, and 6 days at 80°F. Virus was usually demonstrated less frequently in cores taken 3 in. from the initial core than in the other cores.

Symptoms developed only in control roots that received cores of infected tissue. In this case, 54.7% of the inoculated roots developed symptoms. A comparison of this percentage of "takes" with those of bioassay cores cut 1, 2, and 3 in. suggests a higher virus concentration in cores taken directly from infected roots.

The influence of necrotic tissue in the inoculum core on virus movement was also tested. Cores of tissue with and without necrosis were cut from infected roots. Ten roots were inoculated with each type of inoculum and incubated 30 days at 80°F in steamed soil. Bioassay cores were cut 1 in. from the initial cores and inserted into healthy roots. After 4 months in the greenhouse, symptoms developed in 6 bioassay roots testing initial inoculations with necrotic cores and in 3 testing initial inoculations with cores free of necrotic tissue. The data indicate a greater virus concentration in inoculum tissues showing necrosis, which suggests the variability that may result from taking inoculum cores at random from diseased roots.

Influence of inoculum source.—Cores of tissue removed from infected roots are often devoid of necrotic tissue. The observation of Aycock and Hughes (2) that such cores from symptomless carriers are less likely to infect healthy roots than are Porto Rico cores containing necrotic tissue suggested that the presence of a lesion in the inoculum core may be related to disease transmission. Cores of infected tissue with and without necrosis were compared as sources of inoculum in a replicated experiment with 3 types of inoculum cores: 1) infected tissue with necrosis, 2) infected tissue without necrosis, and 3) uninfected tissue. The variation in size and shape of the cork lesion made it impossible to standardize the volume of necrotic tissue in each core of treatment 1. A total of 80 roots were inoculated with each type of inoculum, and the inoculated roots were divided into groups of 10 to provide 4 replications at 2 incubation temperatures (75° and 87°F). Inoculated roots were incubated in compartmentalized metal boxes that were initially humidified to provide favorable conditions for graft union and phellem development. After 46 days, all inoculated roots were sliced and examined for necrotic lesions.

More inoculated roots developed symptoms when the inoculum core contained necrotic tissue than when the inoculum core was free of necrotic tissue (Table 2). The tabular data are based on those roots in which the core of inoculum was alive at the time of examination. Survival of inoculum cores was poorer

TABLE 2.—*How incubation temperature and necrosis in inoculum cores influenced internal cork symptoms in Porto Rico roots 46 days after core-graft inoculation*

Inoculum core	Incubated at 75°F [a] No. [b]	% [c]	Incubated at 87°F [a] No. [b]	% [c]
Infected tissue with necrosis	19/34	57.2	17/34	51.0
Infected tissue without necrosis	6/40	15.0	5/26	17.5
Uninfected tissue	0/39	0.0 [d]	0/27	0.0 [d]

[a] Temperature in this chamber fluctuated ± 4°F from this average.
[b] Numerator, roots with symptoms; denominator, roots with living cores of inoculum.
[c] LSD at .01 = 33.3%.
[d] These values not included in statistical analysis.

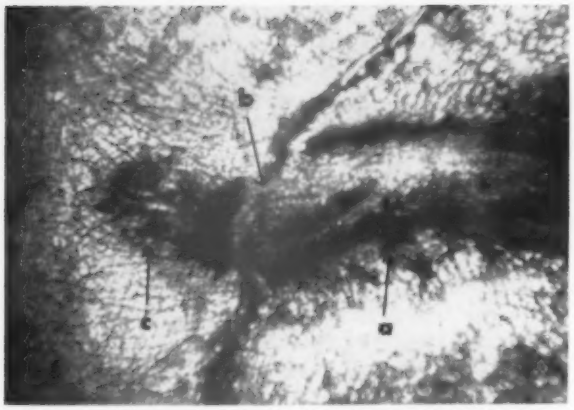

FIG. 1. Movement of internal cork virus from core containing lesion to adjacent healthy tissue as evidenced by the development of necrosis in inoculated root tissue. a) lesion in core tissue; b) graft union; c) lesion in root. ×60.

at the higher temperature. The proportions of roots developing lesions were the same at the 2 incubation temperatures. No symptoms developed in roots "inoculated" with uninfected cores.

The lesions in inoculated roots were generally small, and immediately adjacent to the graft (Fig. 1) or only a few mm therefrom. Most lesions were 1–4 mm in diameter. A majority of the roots with symptoms contained a single lesion, but some contained 2 or 3.

DISCUSSION.—The core-graft method of inoculating sweetpotato roots has a serious limitation: failure of some grafts to survive. For incubation periods of 4–6 weeks this problem is generally not serious, but many grafts decay in incubation periods of 4–6 months. Graft failures are attributable to infection by various root-rotting fungi; improper aeration, relative humidity, and temperature following inoculation; and, possibly, an incompatibility between core tissues and those of the inoculated root. Most important in these studies, it is believed, were infection of the cores by rotting fungi and failure to maintain proper conditions for suberization and graft union.

In the studies on virus movement at different temperatures, about 50% of the grafts survived incubation for 4–5 months. This was true for the 64 controls inoculated with infected tissue: 29 cores survived the incubation period, and 35 roots developed internal cork symptoms. Some grafts developed unions within the inoculated roots and transmitted the virus before the cores decayed. For this reason, the data in Table 1 are based on surviving roots rather than just the roots containing living grafts.

Some grafts also failed in the experiment comparing cores with and without necrotic tissue as sources of inoculum (Table 2). Most failures occurred at the higher incubation temperature. Because of the shorter incubation period and the possibility of different concentrations of virus in the 2 types of inoculum, it was believed better to compare symptom development only in roots exposed to the virus in the cores throughout the incubation period. The data therefore are based on only the roots with living cores.

Virus movement was more rapid at 80°F than at lower temperatures, although the incidence of symptoms later developed in bioassay roots tended to be less. A number of factors may be involved. However, the particulate movement of virus within the suscept as postulated by Samuel (9) and supported by Kunkel (3) appears most tenable. In certain instances these investigators found that tobacco mosaic virus was detected distant from the inoculation site although intervening tissue remained free of virus. At 80°F, the physiological processes within the root proceed at an accelerated rate, as evidenced by sprout production. The accelerated physiological processes, including translocation, may enhance virus movement through the root while minimizing any virus concentration gradient, which could distribute the virus more extensively at the expense of concentration around the inoculation.

The more rapid development of symptoms in roots inoculated with cores containing necrotic tissue suggests that this type of inoculum may contain a greater virus concentration. Bioassay cores removed 1 in. from inoculation sites were more likely to induce symptoms in bioassay roots if the original core contained an internal cork lesion. These data emphasize the desirability of using this type of tissue in core-graft inoculations. The variable results obtained in previous studies may be due, in part, to the indiscriminant use of diseased tissue with or without lesions.

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THE INFLUENCE OF LIGHT AND TEMPERATURE ON THE FORMATION OF INFECTION-TYPE STRUCTURES OF PUCCINIA GRAMINIS VAR. TRITICI ON ARTIFICIAL SUBSTRATES[1]

Robert G. Emge[2]

SUMMARY

Urediospores of *Puccinia graminis* Pers. var. *tritici* (Eriks. & E. Henn.) Guyot form specialized structures during the infection process. Germinated spores were induced to form these on artificial substrates by proper adjustment of environmental conditions. Under optimal conditions of sunlight at 2000–5000 ft-c, temperatures of 80–85°F and a saturated atmosphere, appressoria-like structures formed in 2–3 hours, penetration pegs in 3–4 hours, and vesicles in 4–6 hours. When this period was followed by 16–18 hours of darkness at 85 ± 2.5°F in a saturated atmosphere, formation of vesicles was completed and infection hyphae developed. Conditions above 9000 ft-c and 88°F inhibited the formation of the infection-type structures. If any one factor deviates sufficiently, development and/or differentiation stops and does not resume if the factors are brought back in balance.

INTRODUCTION.—During the infection process, urediospores of *Puccinia graminis* Pers. var. *tritici* (Eriks. & E. Henn.) Guyot form several specialized structures that are apparently necessary for the organism to gain entry and establish itself within the host. These structures are the appressorium, penetration peg, substomatal vesicle, infection hyphae, haustorial mother cell, and haustorium. Sharp et al (5)[3] have shown that the temperature requirements for germination are not the same as those for the formation of infection structures; a single optimum temperature for the complete infection cycle, therefore, does not exist. It has been demonstrated (5) that the infection of wheat plants by *P. graminis* var. *tritici* is materially increased by temperatures that reach 85°F during the last 2 hours of the dew-period (length of time during which free water is present). A similar effect was shown from sunlight in excess of 500 ft-c during this period.

Infection-type structures observed by other investigators (1, 2, 3, 4, 6) have been obtained *in vitro* on specialized surfaces with chemicals and nutrients added to the substrates. The effects of light and temperature were not clearly defined in these investigations. It has since been found that light and temperature play a very important role in the formation of these structures *in vitro* and on the host, and that they can be induced to form in the absence of the host without the benefit of specialized substrates or any added nutrients.

MATERIALS AND METHODS.—The substrates used in this study were acid-washed filter paper (Whatman No. 30), Cellophane dialysis membrane, cellulose membrane filter (Millipore Filter) on the surface of 1% water agar, and 1% water agar itself. Both Syracuse dishes and open Petri plates were used. Hydrated urediospores of *P. graminis* var. *tritici* were dusted on the substrates and germinated 2 hours in the dark at 70–75°F (Fig. 1A), then exposed 6 hours to various light and temperature conditions in a saturated atmosphere, and then 16–18 hours of darkness at several temperatures in a saturated atmosphere. Infection-type structures developed in the normal sequence for this organism.

RESULTS.—The greatest number of the infection-type structures occurred when the germinated spores were exposed to 6 hours of sunlight at 2000–5000 ft-c and temperatures of 80–85°F, followed by 16–18 hours of darkness at 85±2.5°F. Under these conditions, 80% of the germinated spores formed appressoria-like structures in 2–3 hours (Fig. 1-BCD). With germinated spores exposed to these conditions, elongation of the germ tube stops and differentiation takes place. Two hours after the beginning of exposure to these optimal conditions, a slight swelling occurs at the distal end of the germ tube and the protoplasm begins to migrate there from the germ tube (Fig. 1B). This is the initiation of the appressorium. In another hour, the swelling is complete and the protoplasm has concentrated in the tip (Fig. 1C). After 5 hours' exposure, penetration pegs had formed on 60% of the appressoria studied. Fig. 1D shows a well-developed appressorium (a), and a septum (b) between germ tube and appressorium. Also shown is the initiation of a penetration peg (c), observed after 4 hours of exposure. Development of the infection structures overlap in relation to time. This is illustrated in Fig. 1E, where 3 appressoria and 2 penetration pegs are shown in different stages of development after 4 hours of optimal conditions. Two of the appressoria are well developed (a); 1 is in an early stage of development (e); the septum (d) between the germ tube and appressorium is clearly defined. The appressorium was considered fully developed only after this septum was formed. Also readily seen are an early stage of the penetration peg (b) and a later stage (c). After 5–6 hours of exposure, the tips of 90% of the penetration pegs had begun to enlarge (Fig. 1F). The protoplasmic material flowed from the appressorium through the peg and concentrated in this swelling (b); thus the substomatal vesicle was formed. Fig. 1F illustrates a substomatal vesicle in

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[3] The author wishes to acknowledge valuable discussions with the authors of that paper.

an early stage of development. The septum (a) is shown between the appressorium and germ tube. In this instance, the structure was observed after the germinated spores had been exposed to about 5½ hours of optimal light and temperature conditions. At the end of 6 hours most of the vesicles had reached

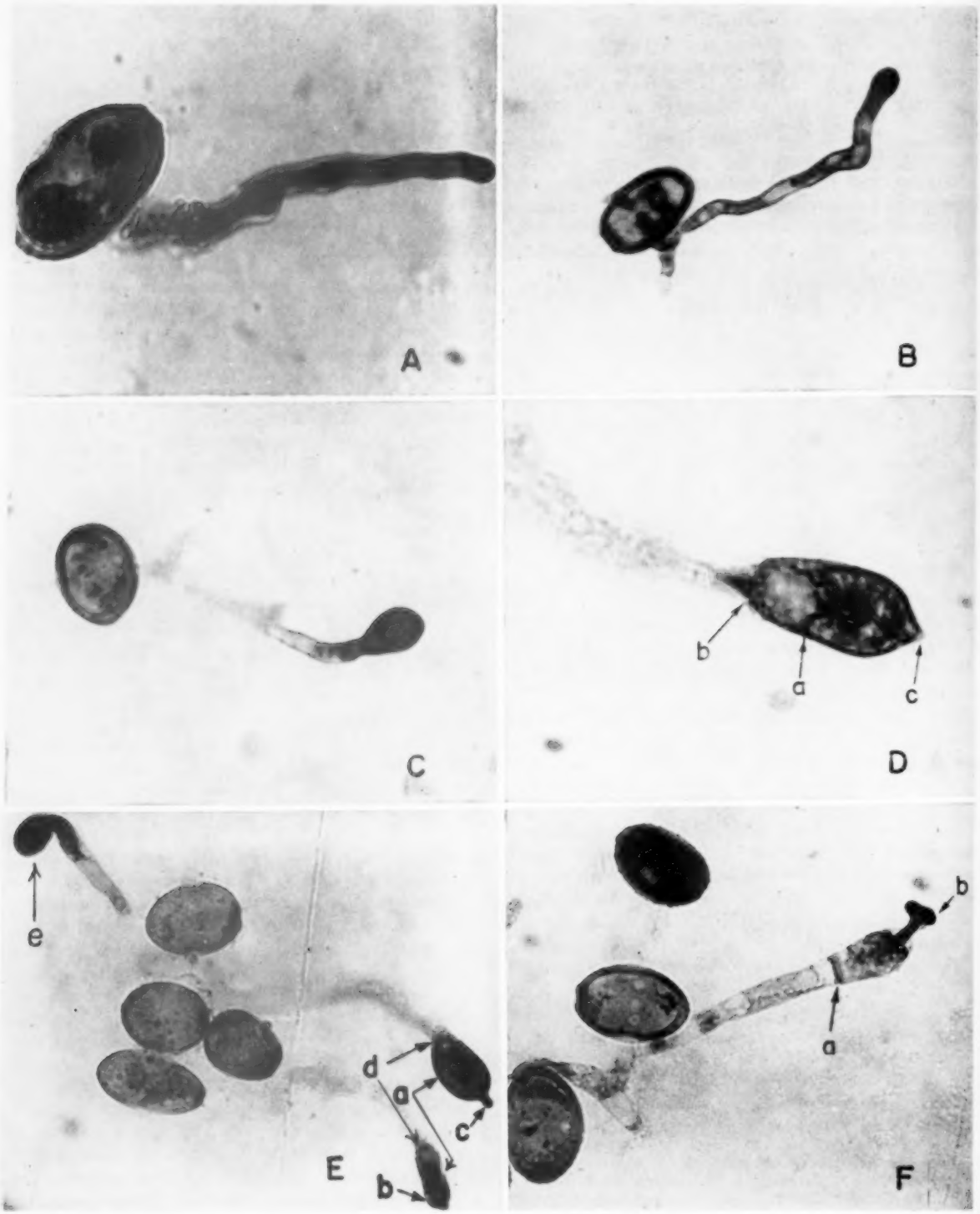

FIG. 1. A) Germinated urediospore. B) Early stage of appressorial formation. C) Later stage of appressorial formation. D) Fully developed appressorium. E) Several stages in the development of the appressoria and penetration pegs. F) Early stage in the development of the substomatal vesicle. (U. S. Army photographs.)

full development (Fig. 2A). The septa between the germ tube and the appressorium (a) and between the appressorium and vesicle (b) are shown. The vesicle (c) is considered fully developed when this latter septum is formed. The infection hyphae (Fig. 2B) develop from this structure. After 16–18 hours in the dew chamber that succeeded the 6 hours of optimal light and temperature conditions, infection hyphae (Fig. 2-BCD) were observed on all the vesicles in all plates maintained at 85±2.5°F. Fig. 2B shows the infection hyphae in an early stage (c), and Fig. 2C and 2D show progressively later stages, at (c) and (b), respectively. The last is considered fully developed.

Conditions other than those described will also induce this organism to form infection-type structures, e.g. 1) sunlight at 2000–5000 ft-c and temperatures of 75–80°F, 2) darkness and temperatures of 85± 2.5°F, and 3) fluorescent white light, at 1000 ft-c and temperatures of 78–81°F. Under such conditions the formation of infection-type structures proceeded at a slower rate, and only about half as many structures were observed as under optimal conditions. Sunlight above 9000 ft-c or temperatures of 88°F or above inhibited their formation.

Discussion.—The effect of the environmental factors tested appears to be continuous, in that if any one of the factors deviate sufficiently from those that induce the formation of the structures, the development

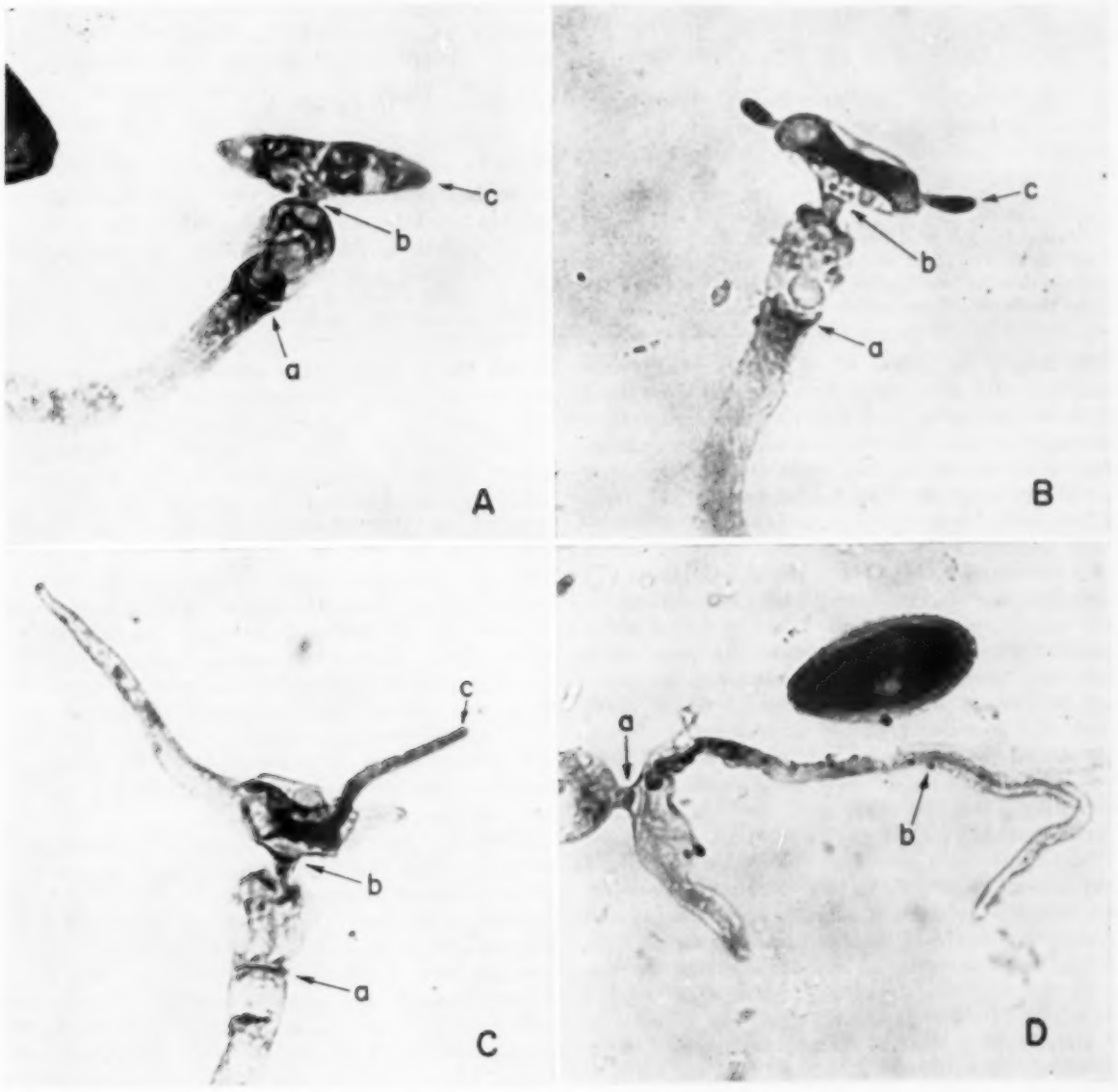

Fig. 2. A) Fully developed substomatal vesicle. B) Substomatal vesicle with early stage of infection hyphae. C–D) Substomatal vesicles with well-developed infection hyphae. (U. S. Army photographs.)

and/or differentiation will stop. If these factors are subsequently returned to optimal, development will not resume.

The results indicate that light and temperature are dominant in the formation of the infection structures. When germinated spores were exposed to the proper combinations of light, temperature, and moisture, these structures formed on the substrates used and in absence of the host.

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CHEMICAL CONTROL OF CEREAL RUSTS. I. PROTECTIVE AND ERADICATIVE CONTROL OF RYE LEAF RUST IN THE GREENHOUSE WITH VARIOUS CHEMICAL COMPOUNDS[1]

Harry L. Keil, Hans P. Frohlich, and John O. VanHook[2]

SUMMARY

In laboratory and greenhouse studies, many compounds were found with protective properties against the rye leaf rust fungus. A limited number of these also had eradicative properties. Among those having the capacity to stop or suppress the development of rust after infection took place, the nickel salt amine complexes were the most promising. In these the eradicative property is due to the nickel present in the molecule, and the protective property is associated with both the amine and nickel.

The search for means of controlling cereal rusts continues. For some time plant breeders have been diligently producing new wheat varieties that show resistance to rust; but when a supposedly resistant wheat is introduced, it is not many years before a new race of rust infects it. This has happened many times, as both farmer and scientist can attest.

The farmer cannot stand several years of severe losses while the geneticist hybridizes, field tests, and multiplies seed stocks for a new resistant variety. A good example of this is severe losses by durum wheat growers during the last 4-6 years. In some areas, losses have been so bad that farmers found it impractical to attempt to harvest the crop. These heavy losses, and mounting losses in the soft-bread wheats, have stimulated interest in finding a chemical control. Borlaug (1) and McNew (5), as well as others, have pointed out the great need for chemical compounds that will control rust of wheat.

Many researchers have been able to protect wheat from rust infection with various chemical compounds, such as sulfur or zineb, applied frequently before the infection is established. If the plant is protected from seedling stage to maturity, control is almost perfect, though the program relies principally on protection and is expensive and time-consuming.

The farmer would prefer to delay treatment until infection has taken place. This means a chemical is needed that will eradicate the rust after it is established. Both Livingston (4) and Hotson (3) have reported on such compounds within recent years. Livingston showed that good control was obtained with calcium sulfamate and phenyl hydrazine. Under both greenhouse and field conditions these compounds could effectively suppress and even stop the disease after infection had taken place. Hotson, with sulfa drugs, showed essentially the same type of action. Of the compounds he tested, the most effective in suppressing rust development of stem rust of wheat was the sodium salt of sulfadiazine. These compounds as well as others are currently being field-tested by several investigators.

MATERIALS AND METHODS.—Soon after starting our screening program for rust fungicides, it became evident that many compounds are effective protectants against cereal rusts. The difficult compounds to find, however, are those that stop rust development or at least suppress it after infection has taken place.

In preliminary tests, results were similar with rye leaf rust, *Puccinia rubigo-vera* (DC) Wint. f. sp. *secalis* (Ericks.) Carleton, and with wheat stem rust (Race 15B), *Puccinia graminis* Pers. f. sp. *tritici* Eriks. & E. Henn. Under our greenhouse conditions,

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rye leaf rust is more easily cultivated than wheat stem rust, and was therefore used as the test organism for the studies reported herein. Two types of tests were conducted: 1) protective, and 2) eradicative.

Uredospores of *P. rubigo-vera* f. sp. *secalis* were diluted with distilled water so that the final suspension contained 25,000 spores per ml. (The binomial *Puccinia recondita* Rob. ex Desm. was recently suggested for this fungus (2), by Cummins and Caldwell.) This suspension also contained 0.1% sucrose and 0.001% potassium citrate, to aid germination, and 0.0025% Triton NE (an alkyl aryl polyether alcohol), to improve spore dispersion and wetting of the foliage. Seedlings of Balbo rye 10–12 days old, 50 plants per 4-in. pot, were trimmed to a height of 4.5 in. and inoculated by spraying on 7 ml of spore suspension per pot.

Immediately after inoculation the plants were placed in an illuminated incubation chamber and held there for 24 hours at 100% relative humidity and 68°F. The plants were then returned to the greenhouse bench for the remainder of the test. Despite an attempt to control greenhouse temperatures at 65–70°F, the temperature on occasion reached 80°F or more during the daytime, especially during sunny periods.

In the protective tests the chemicals were sprayed onto the plants with a DeVilbiss atomizer (#152) under pressure of 25 psi. Plants were placed on a turntable and sprayed to the point of run-off, which took about 20 ml per pot. The plants were then allowed to dry on a greenhouse bench (usually taking less than 2 hours), inoculated, and incubated 24 hours. After 11 additional days on the greenhouse bench they were evaluated for rust infection.

In the eradicative tests, plants at the fleck stage (5 days after inoculation) were sprayed to run-off point, held 7 days on the greenhouse bench, and evaluated for rust infection.

All treatments were replicated 2–3 times. To facilitate wetting of the foliage, either Triton B-1956 (a modified alkyd resin, Rohm & Haas Co.) or Triton X-114 (an alkyl aryl polyether alcohol, Rohm & Haas Co.) was used at a concentration of 1:4000. The first was used in the early tests; then it was found that Triton X-114 gave better wetting of certain compounds.

Each pot of 50 plants was rated on a 0–5 scale: 0 = no pustules; 0.25 = 1–5 pustules; 0.5 = 6–20 pustules; 0.75 = 21–50 pustules; 1 = 51–100 pustules; 2 = 101–200 pustules; 3 = 201–500 pustules; 4 = 500–1000 pustules; and 5 = >1000 pustules.

RESULTS.—Of the 200 compounds tested up to June, 1956, many showed protective properties but relatively few eradicated rye leaf rust after infection took place. Two types of eradication were observed: 1) no reduction in the number of pustules or sori erupting, but the developing uredospores changed from dark brown to light yellow; 2) a reduction in the number of erupting or open pustules, with or without some change from brown to yellow in the pustules producing uredospores. With complete eradication, the pustules turned black and dried up. In inoculation studies, spores that became yellow did not infect new plants; dark brown spores did. From the eradicative point of view, the following chemical types were of particular interest:

Metal amine complexes.—It was discovered early in the screening program that rye leaf rust could be controlled by certain metal amine complexes. In eradicative studies conducted at that time the RH-1 (convenience code used herein for bis[*N*-(2-hydroxyethyl)dodecylmethylbenzylamine] nickel (II) chloride), applied to plants at the fleck stage, prevented pustule development without injuring the plants objectionably. In the few cases where pustules developed, the uredospores turned yellow and failed to infect new plants in inoculation studies. This same compound also proved very effective in protective studies.

What part is played by the component parts of RH-1? Preliminary studies undertaken to answer this question indicated that the eradicative property of RH-1 (and certain other nickel amine complexes) is due to the nickel part of the molecule, and that the protective property is associated with both the amine and nickel parts. The cupric chloride, cadmium chloride, nickel chloride, and zinc chloride complexes derived from the amine *N*-(2-hydroxyethyl)dodecylmethylbenzylamine were each effective as protectants, but only the nickel analog gave effective eradication at non-phytoxic dosages.

Typical data obtained in both protective and eradicative tests with 7 nickel chloride amine complexes are presented in Table 1. All may be considered very good protectants, with the index never going over 0.75 (meaning that the average number of pustules per plant was no greater than 1; in most cases it was a fraction of 1). In the eradicative tests, RH-6 (bis[*N*-(2-hydroxyethyl)dodecylbenzylamine] nickel (II) chloride) showed the best control. However, the other nickel chloride amine complexes also gave good eradication of the rust, rating 1.0 or lower at the 1-lb. level. At the ½-lb. level the compounds ranked from best to poorest (0.75–4.0) as follows: RH-6; RH-2 (bis[*N*-(2-hydroxyethyl)5,5,7,7-tetramethyl-2-octenylamine] nickel (II) chloride); RH-4 (bis[*N*,*N*-di(2-hydroxyethyl)dodecylmethylbenzylamine]' nickel (II) chloride); RH-1; RH-3 (bis[*N*,*N*-di(2-hydroxyethyl)-5,5,7,7-tetramethyl-2-octenylamine] nickel (II) chloride); RH-7 (bis-imidazoline made from Emory Dimer acid and ethylenediamine complexed with nickel (II) chloride); and RH-5 (bis[di(dodecylmethylbenzyl)-2-hydroxyethylamine] nickel (II) chloride). None of the compounds gave excessive injury at the tested concentrations, except RH-4, which showed moderate injury at the 1-lb. level.

Dinitro-1-methylheptylphenol derivatives.—Another group of compounds found to give good control of

TABLE 1.—*Protective and eradicative control of rye leaf rust with various chloride amine complexes*

Compound [b]	Code no.	Pounds per 100 gal. water	Rust index [a] Protectant	Rust index [a] Eradicant	Injury
Bis[*N*-(2-hydroxyethyl) dodecylmethylbenzylamine] nickel (II) chloride	RH-1	1	0.0	0.9	Very slight
		0.5	0.1	2.0	None
Bis[*N*-(2)-hydroxyethyl) 5,5,7,7-tetramethyl-2-octenylamine] nickel (II) chloride	RH-2	1	0.4	0.9	Very slight
		0.5	0.75	1.5	None
Bis[*N,N*-di (2-hydroxyethyl) 5,5,7,7-tetramethyl-2-octenylamine] nickel (II) chloride	RH-3	1	0.25	0.6	Slight
		0.5	0.5	2.0	Very slight
Bis[*N,N*-di (2-hydroxyethyl) dodecylmethylbenzylamine] nickel (II) chloride	RH-4	1	0.1	0.6	Moderate
		0.5	0.4	1.5	Slight
Bis[di (dodecylmethylbenzyl)-2-hydroxyethylamine] nickel (II) chloride	RH-5	1	0.0	1.0	None
		0.5	0.25	4.0	None
Bis[*N*-(2-hydroxyethyl) dodecylbenzylamine] nickel (II) chloride	RH-6	1	0.0	0.25	Slight
		0.5	0.0	0.75	Very slight
Bis-imidazoline made from Emory Dimer acid [c] and ethylenediamine complexed with nickel (II) chloride	RH-7	1	0.25	0.9	None
		0.5	0.5	2.5	None
Untreated			5.0	5.0	None

[a] Average rust index based on readings made on 2 pots (50 plants per pot). Ranked according to the following: 0 = no pustules, 0.25 = 1-5 pustules, 0.5 = 6-20, 0.75 = 21-50, 1.0 = 51-100, 2.0 = 101-200, 3 = 201-500, 4 = 501-1000, 5 = >1000 healthy pustules per pot.

[b] Triton B-1956 (a modified alkyd resin, Rohm & Haas Co.) added at rate of 1:4000.

[c] A polymerized fatty acid obtained from Emory Industries, Inc.

rye leaf rust are the dinitro-1-methylheptylphenol derivatives. Many of the compounds in this group show good protective properties, and some eradicate the disease after it becomes established. The type of eradication usually observed with these is different from that found with the nickel compounds. These chemicals, in eradicative tests, usually allow the pustules to develop after treatment at the fleck stage. The uredospores in many of these pustules turn yellow, whereas untreated pustules produce dark brown spores. Several attempts to produce infection with the yellow spores failed; the dark brown spores always caused infection on newly inoculated plants. The change from dark brown to yellow usually takes place at weak concentrations, such as 0.5 lb. or lower. With higher concentrations the flecks in the eradicative tests often turn white and dry up.

Two typical members of this group of compounds are 2-(1-methylheptyl)-4,6-dinitrophenyl crotonate and isomers (Karathane) and dimethylamine salt of dinitro-1-methylheptylphenyl. In protective tests both showed perfect control at 0.5 lb. per 100 gal. In eradicative studies, Karathane at the same level failed to show any reduction in number of pustules, but about 95% of the developing pustules produced yellow uredospores. The dimethylamine salt, on the other hand, showed a striking reduction of developing pustules (150 pustules per pot of 50 plants, compared to over 1000 in check pots). This reduction may, however, have been due to injury, since severe damage of the leaf tissue was noted at the dosage of 0.5 lb. per 100 gal.

Other compounds.—Dithiocarbamates and derivatives such as disodium ethylenebisdithiocarbamate (Dithane A-10) and ethylenebisthiuramtrisulfide (Lo-738) showed perfect control in protective tests. Eradicative results were different: Dithane A-10 showed some reduction in number of pustules, but this apparently was influenced by the objectionable foliage injury noted at the test concentrations. Lo-738 gave variable results, with hardly any detectable reduction in number of pustules in some eradicative tests, and quite pronounced reduction in others. These results were not confounded with injury, unlike those with Dithane A-10.

A fourth group of compounds showing evidence of control in rust studies is the organic sulfites. A typical member of this group, 2-chloroethyl 4-chlorophenyl sulfite, showed excellent control in the protective trials at 1-lb. and 0.5-lb. per 100 gal. In the eradicative studies, the number of pustules was significantly reduced at the 1-lb. level but not at the 0.5-lb. level. Again eradicative action appeared correlated with injury. Moderate to severe injury was observed at the 1-lb. level, and very little at the 0.5-lb. dosage.

Of the compounds showing both protective and eradicative properties, the most promising appear to be the nickel-containing compounds. This was borne out in wheat-rust field tests conducted during 1956 in Canada by Peturson, Forsyth, and Lyon (6).

DISCUSSION.—The nickel salt amine complexes appear to have, in addition to protective properties, desirable eradicative properties as well. These chemicals gave pronounced eradication of rust at concentrations not causing objectionable plant injury. Data from the protective and eradicative tests indicate that the protective activity of these nickel salt amine complexes is associated with both the amine and nickel parts of the molecule, and the eradicative or chemotherapeutic activity with the nickel part.

Most compounds other than those containing nickel appear to have a direct correlation between the rust

eradicative property and phytotoxicity. This seems to be especially true where a reduction in number of erupting pustules takes place. When non-phytotoxic concentrations are used there is no reduction in number of pustules as indicated by the index system. Most nickel compounds tested, however, do show a substantial decrease in number of pustules even at non-phytotoxic concentrations, suggesting a different kind of eradicative action.

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CHEMICAL CONTROL OF CEREAL RUSTS. II. CONTROL OF LEAF RUST OF WHEAT WITH EXPERIMENTAL CHEMICALS UNDER FIELD CONDITIONS[1]

B. Peturson, F. R. Forsyth, and C. B. Lyon[2]

SUMMARY

Thirteen chemicals were tested for rust control in field plots at Winnipeg. Effective control in this field trial was provided by 4 nickel salt amine complexes, all of which appeared to possess both protective and eradicative properties: RH-6, bis[*N*-(2-hydroxyethyl)dodecylbenzylamine] nickel (II) chloride; RH-1, bis[*N*-(2-hydroxyethyl)dodecylmethylbenzylamine] nickel (II) chloride; RH-2, bis[*N*-(2-hydroxyethyl)dodecylamine] nickel (II) chloride; and RH-3, bis[*N*,*N*-di(2-hydroxyethyl)dodecylamine] nickel (II) chloride. Nickel was a component of all compositions that appeared to have good rust control properties.

Stem rust (*Puccinia graminis* Pers.), leaf rust of wheat (*Puccinia rubigo-vera* (DC.) Wint.), and crown rust of oats (*Puccinia coronata* Cda. f. sp. *avenae* Eriks. & E. Henn.) occur annually on cereals in North America in most grain-growing areas. In some years they appear in epidemic form, damaging crops in varying degree on millions of acres in Canada and the United States. Destructive epidemics occurred in parts of Western Canada and in parts of the upper Mississippi Valley in 1904, 1911, 1916, 1920, 1923, 1925, 1927, 1935, 1938, 1953, and 1954. The epidemics of 1916 and 1935 were particularly severe, completely destroying wheat on thousands of acres.

Owing to these frequent and severe rust losses, there has been much interest in rust control measures during the past fifty years. Control attempts have included plant breeding and cultural methods, and applications of fungicidal sprays and dusts.

From 1924 to 1939, experimental work was carried out in North America to determine the feasibility of controlling cereal rusts by chemical means. Sulphur was the chief fungicide used. Bailey and Greaney (1), Greaney (4), Kightlinger (6), and Stakman and Person (8) all found that timely applications of sulphur dust several times during the season controlled cereal rusts.

After 1939, when rust-resistant cereal varieties became available, interest diminished in chemical methods of rust control, and little experimental work was done on rust-control chemicals during the next thirteen years. But in 1952, when races of rust pathogenic to the predominant commercial varieties (Thatcher, Redman, Regent, and Apex) became prevalent, interest in this method of control was renewed. Many new chemicals for rust control have been tested during the past few years by Brentzel (2), Livingston (7), Wallen (9) and Forsyth and Peturson (3). Although several fungicides (zineb, maneb, ferbam, Phygon, Thioneb, Sulforon, Acti-dione, nabam plus zinc sulphate, and calcium sulfamate) controlled cereal rusts if applied several times during the growing season, none was of sufficient promise to be recommended for rust control as a general farm practice. The chief shortcomings of many of these materials were their high cost and the number of applications (in some cases 4) required for satisfactory control. Of the better rust control fungicides, nabam plus zinc sulfate was much the cheapest. Its cost per acre per application, for materials and expense of application, amounted to about $1.55, and at least 3 applications were needed for good rust control.

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Certain defects and the high cost of currently available rust control fungicides have led the authors, as well as many other workers, to search for better and cheaper materials. Promising rust control chemicals are tested as they become available.

In 1956, 13 chemicals were obtained that had shown some promise for rust control in greenhouse tests carried out by the Rohm & Haas Company (5). These chemicals were tested for control of leaf rust of wheat in a field test at Winnipeg in 1956. This paper deals with the results of that test.

MATERIALS AND METHODS.—For convenience, the chemicals used are herein designated as follows: Dithane A-10, RH-10, RH-11, Lo-738, RH-7, RH-1, RH-12, RH-2, RH-3, RH-4, RH-5, RH-6, and Karathane EC.[3] Their chemical compositions are given in Table 1.

Each chemical was used at 2 concentrations: 2 and 4 lb./A. in spray form. Two sprays were applied during the season: the first when the plants were coming into the heading stage and carried an average leaf-rust infection of 3–5% on the lower leaves; the second 2 weeks later. The first rust reading was taken 1 day after the second spray application; the second reading 6 days later.

The experimental area, in a late planting (June 13), was planted specifically for the cross-comparison of these experimental rust fungicides; the test plants were of the variety Thatcher, grown in rod-row plots. Each plot consisted of 3 rows spaced 1 ft. apart. Each treated plot was separated from adjoining treated plots by 3 rod-row buffer plots of untreated Thatcher wheat. The experimental field was divided into 4 blocks, each consisting of 2 equal sub-blocks containing an untreated check and 13 plots, each sprayed with one of the test materials. In each block the treated plots were sprayed at the rate of 2 lb./A. in one of the sub-blocks, and 4 lb./A. in the other. All materials were applied in water sprays at 55 gal./A.

The analysis of variance method was used in evaluating the significance of the data obtained. In these analyses, all percentage readings were transformed to degree angles.

RESULTS AND DISCUSSION.—Although sown late, the plants made good growth and reached the heading stage by August 7, when the first spray was applied. Leaf rust infections, developing uniformly throughout the experimental field, had reached an average severity of 3–5% on the lower leaves. Many further infections were doubtless developing in the leaves at that time. The second application was made on August 21, 14 days after the first application. During the interval, infection in the check plots increased to 67%.

Leaf rust increased substantially in the plots treated with 8 of the test chemicals at the 4-lb. rate (RH-11, RH-12, Dithane A-10, RH-5, Lo-738, Karathane, RH-7, and RH-10), with final readings ranging from 39 to 80% (Table 2). Four of the chemicals (RH-6, RH-1, RH-2, RH-3) materially retarded leaf rust development; average infections in the plots to which they were applied at the 4-lb. rate ranged from 6 to 15%. Two of the chemicals (RH-1 and RH-6) controlled leaf rust almost completely; during the 14-day period between the 2 applications there was practically no rust increase in the plots.

On August 28, 3 weeks after the first application and 1 week after the second, a final estimate was made of the amount of leaf rust in the plots. During the week following the second application, rust infection increased with fair uniformity (15–20%) in all plots except those treated with RH-1 and RH-6 (Fig. 1), which had respective increases of about 10 and 3%. The final percentages of leaf-rust infection in treated and check plots are given in Table 2. The table also gives the average degree angle transformations derived from the rust percentages. The table shows that 4 of the chemicals—RH-6, RH-1, RH-2, and RH-3—gave good leaf rust control at the higher concentration, and that the plots treated with these materials had significantly less rust than the check plots and plots treated with the other chemicals. RH-6 and RH-1 also gave good rust control at the lower concentration, but RH-2 and RH-3 were inferior to RH-6 and RH-1 at this concentration. Much inferior were all the other chemicals, though some gave partial control.

The 4 chemicals RH-6, RH-1, RH-2, and RH-3 all arrested, to a considerable extent, infections that had taken place before these chemicals were applied. About 3 days after application of these chemicals a dark ring of discolored tissue formed around the infection site of the pustules, and further development

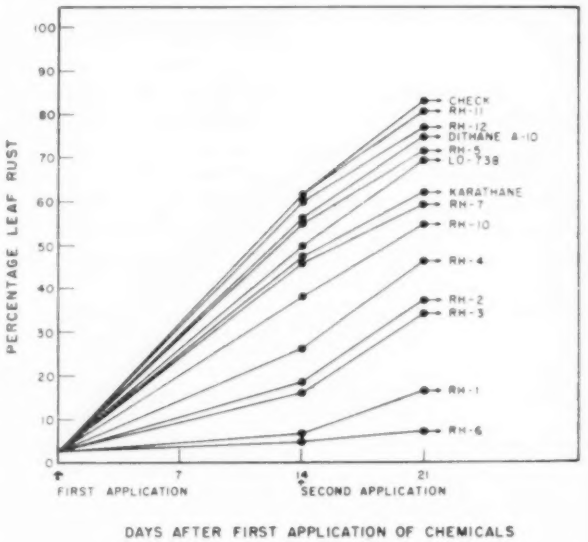

FIG. 1. Percentage of leaf rust of wheat in field plots of Thatcher treated with 13 rust-control chemicals (averages for 2 concentrations of each chemical).

[3] The chemicals were supplied by the Rohm & Haas Company.

TABLE 1.—*Chemical composition of 13 chemicals tested at Winnipeg for cereal rust control*

Code designation	Chemical formula
Dithane A-10 *	Spray-dried disodium ethylenebis[dithiocarbamate]
RH-10 *	$(CH_3)_2NH_2 \cdot OC_8H_2$ capryl $(NO_2)_2$
RH-11 *	2-chloroethyl-4-chlorophenyl sulfite
Lo-738	ethylenebisthiuram trisulfide—75% wettable powder
RH-7 *	$NiCl_2$ complex of the imidazoline from Emory dimer acid and ethylenediamine
RH-1	$Ni[C_{12}H_{25}(CH_3)C_6H_3CH_2NHCH_2CH_2OH]_2Cl_2$—40% solution in ethyl alcohol
RH-12	$Zn[C_{12}H_{25}(CH_3)C_6H_3CH_2NHCH_2CH_2OH]_2Cl_2$—16% solution in ethyl alcohol
RH-2	$Ni[C_{12}H_{25}NHCH_2CH_2OH]_2Cl_2$—25% solution in ethyl alcohol
RH-3 *	$Ni[C_{12}H_{25}N(CH_2CH_2OH)_2]_2Cl_2$
RH-4 *	$Ni[C_{12}H_{25}(CH_3)C_6H_3CH_2N(CH_2CH_2OH)_2]_2Cl_2$
RH-5	$Ni[C_{12}H_{25}(CH_3)C_6H_3CH_2)_2NCH_2CH_2OH]_2Cl_2$—25% solution in ethyl alcohol
RH-6	$Ni[C_{12}H_{25}C_6H_4CH_2NHCH_2CH_2OH]_2Cl_2$—30% solution in ethyl alcohol
Karathane	2-(1-methylheptyl)-4,6-dinitrophenyl crotonate and isomers (50% emulsion)

* 100% active ingredient.

of the pustules was arrested. Many of the infections developing inside leaves when these materials were first applied were, doubtless, also arrested, as indicated by the failure of rust to increase between applications. Numerous other infections would be developing in the leaves at that time, and their failure to develop indicates the eradicative properties of the materials used. Although, statistically, there was no significant difference between RH-6 and RH-1 in effectiveness at the lower concentration, and no significant differences among the 4 best chemicals at the 4-lb./A. concentration, RH-6 seemed to possess slightly better rust control properties than any of the other chemicals tested.

The 13 rust-control chemicals tested in this experiment were appraised solely on their effectiveness in suppressing rust development. Their effect on yield, bushel weight, kernel weight, and grade could not be determined, because of very severe frost damage to the plants on September 9, when the kernels were still quite green.

All of the best rust-control chemicals had one property in common: nickel was a component. It may be that this common component was chiefly responsible for the eradicative and protective properties of these chemicals.

Fig. 1 shows the course of rust development in the plots as indicated by the initial amount of rust and the percentages of rust at the 2 rust readings. The uniform slope of the lines indicates that rust in the check plots and in the plots treated with the less effective chemicals developed at a fairly uniform rate. The flat slope of the lines for the 2 chemicals RH-6 and RH-1 during the 14-day period between applications, as well as visual observations, indicates that these materials possess both eradicative and protective control properties.

TABLE 2.—*Average leaf-rust infection 3 weeks after first application and 1 week after second application in the Thatcher variety of wheat treated with 13 chemicals at 2 amounts to an acre*

Treatment	2 lb./A. Av. % leaf rust	2 lb./A. Rust % transformed to ° angles	4 lb./A. Av. % leaf rust	4 lb./A. Rust % transformed to ° angles
RH-6	10.5	18.7**	5.5	15.5**
RH-1	23.8	28.7**	8.0	16.2**
RH-2	65.0	54.3*	10.0	18.4**
RH-3	55.0	49.3**	15.0	22.4**
RH-4	53.8	46.7**	38.8	38.2**
RH-10	61.3	51.6*	48.8	44.2*
RH-7	75.0	61.1	46.3	42.7**
Karathane EC	62.5	52.5*	63.8	53.1*
Lo-738	76.3	61.0	63.8	53.4
RH-5	78.8	63.2	68.8	61.1
Dithane A-10	78.8	62.8	73.8	59.7
RH-12	80.0	65.1	77.5	61.8
RH-11	83.8	66.2	80.0	63.7
Check	81.3	64.4	83.8	66.2
LSD between means of 2 chemicals (0.5)		12.4		12.4
LSD between means of 2 chemicals (0.1)		16.6		16.6

* Significant at the 5% level.
** Significant at the 1% level.

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DETERMINATION OF THE EFFECTS OF DIFFERENT TEMPERATURES ON UREDIAL INFECTION WITH PUCCINIA GRAMINIS VAR. TRITICI[1]

C. T. Lange, C. H. Kingsolver, J. E. Mitchell, and E. Cherry[2]

SUMMARY

A variable-temperature incubation chamber permits control between 50° and 100°F by regulating temperatures in water bath and cooling coils. An 8° differential between air and water temperature gave the most uniform dew deposition and the best infection. Tests at continuous temperatures during a 20-hour dew with Baart wheat seedlings established optimum infection with *Puccinia graminis* var. *tritici* at 75°, minimum near 60°, and maximum near 85°F.

This investigation was made to determine the critical continuous temperatures during dew for infection by urediospores of *Puccinia graminis* var. *tritici* Erikss. & Henn. A group of new dew chambers, similar to a prototype previously described (6), were used. Proper combinations of incubation time, temperature, and RH (relative humidity), together with viable inoculum and susceptible host, are required for infection success. The literature on infection by rust fungi attacking cereals contains many different recorded incubation temperatures, which the various authors must have either regarded as optimum or used out of necessity. A few of the many references are listed to show the conditions used by various authors: Melhus (5) 61°F at 95% RH for 24 hours; Cherewick (1) 50–59°F; Tervet et al (9) 65°F for 12 hours; Hotson (2) 63°F for 18–20 hours; Stakman and Levine (8) 66.5–70°F; and Peltier (7) 77°F for "form" 3 and 68°F for "form" 9. A more comprehensive review of the literature involved is given by Lange (3).

Recent advances in incubation chamber design have made possible tests of an exact nature, in which the temperature of the microclimate during incubation could be varied within a range at the discretion of the investigator. These dew chambers (3) have been in use at Fort Detrick since March of 1953.

MATERIALS AND METHODS.—*Dew chambers.*—The dew chambers are basically simple rectangular enclosures with interior dimensions of 30 in. square by 54 in. high. The side and back walls, an area of 33.75 sq. ft., are formed of a series of fluid-filled coils (cold plates). The top and front of the chambers are insulated panels of stainless steel. The door, including an observation window, is in the front wall of the chamber. A light is recessed in the center of the top, behind 2 panes of glass with a ½-in. insulating air interstice. The bottom 4 in. of the chamber comprises a controlled-temperature water bath. The air temperature in the chamber is a function of water-bath temperature and temperature of the fluid coolant circulating in the walls. The term "temperature differential," used throughout this paper, is defined as the difference in temperature in degrees F between the air in the dew chamber and the water bath. Air temperature can be controlled at any value between 50° and 100°F, depending on the temperature maintained in the water bath and in the cooling coils. Two chambers are shown in Fig. 1A.

Fig. 2 and the accompanying text explain the operation of the chambers. One large cooling unit supplies refrigerated water for all of the chambers. The cooling effect of this unit in a single dew chamber is accomplished through a heat exchanger. An automatically controlled three-way valve regulates the cooling mechanism for a single chamber. When no cooling is required the valve diverts all of the coolant back to the storage tank, by-passing the heat exchanger. A constant air temperature is maintained by balancing the cooling and heating systems of any one dew chamber. The heating unit, located in the water bath, has 2 elements (100-watt and 250-watt). When cabinet temperatures drop below the control point, the 100-watt heater is energized, and then the 250-watt heater. As the temperature rises again to the control point, the 250-watt heater cuts off first, and then the 100-watt heater. This arrangement affords a more uniform control of the dew chamber air temperature than would a single heater. The water bath is always maintained at a temperature above that of the dew chamber air. Thus free water deposits on the foliage of test plants by condensation rather than by mechanical atomization.

All of the air-temperature sensing elements extend 9 in. into the chamber from the top. These elements are connected to an electronic recording potentiometer, which provides a printed record of the dew-chamber air temperatures during all tests. Temperatures for the different treatments during a 20-hour test were obtained by averaging the temperatures recorded at 30-minute intervals for each cabinet.

Air temperatures of nine dew chambers were adjusted to about 60, 65, 70, 72.5, 75, 77.5, 80, 82.5, and 85°F. Previous investigations of infection temperatures, at 5° intervals from 50 to 90°F, had suggested

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more intensive trials between 60° and 85°F.

Host Plants.—Seeds of Baart wheat (C.I. 1697) were planted in a row across clay pots of 4-in. diameter. Pots of 7- to 10-day-old seedlings were trimmed to 10 primary leaves. These pots were placed with both line of planting and seedling leaves in the same horizontal plane on the metal mesh platform of the dew chambers (Fig. 1B). Ten pots with 10 leaves each were placed in each dew chamber.

Inoculum and inoculation method.—Departure was made from the standard method of settling-tower inoculation used in this laboratory and discussed by Lange (3). To save time, a less refined but sufficiently precise method was used. Twenty mg of urediospores of a specific race were weighed and placed in size-00 gelatin capsules. The capsules were placed in the U-shaped end of a 4-ft. discharge tube, which was inserted vertically into the top of each closed dew chamber through a small access hole. The spores were discharged upward into each chamber by an instantaneous blast of compressed CO_2, which fractured the capsule on a sharp blade and created a cloud of spores with a minimum of clumps. The blast was obtained with a Crossman CO_2-type target pistol threaded to attach to the discharge tube. Deposition of controlled amounts of urediospores on the leaves was satisfactorily uniform. After 20 hours of dew, plants were moved to greenhouse benches maintained at 75 ± 1°F. Pustules were counted 10–14 days after inoculation.

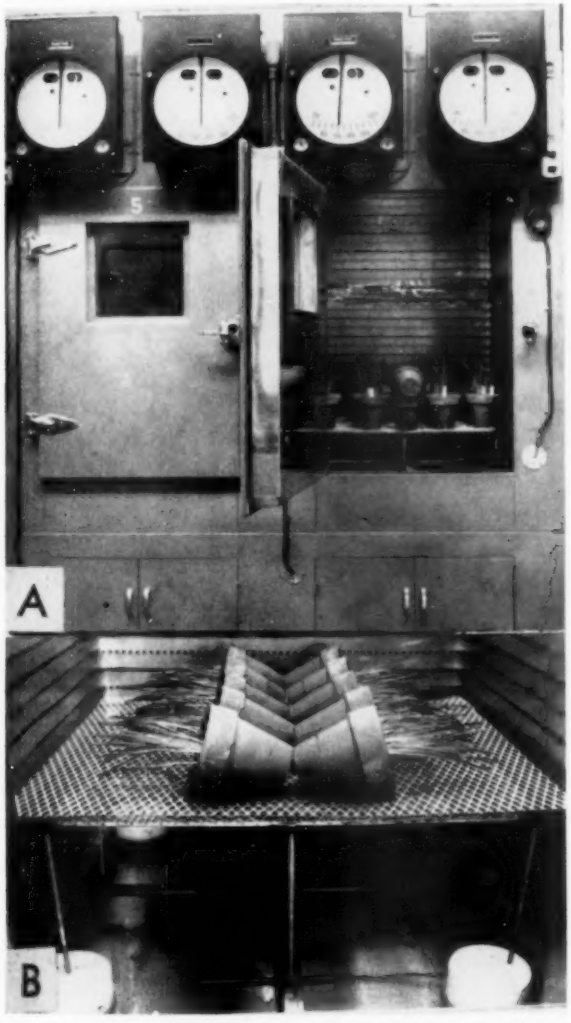

FIG. 1. Variable temperature dew chambers. A) Front view of 2 cabinets. B) Chamber with plant material arranged for inoculation.

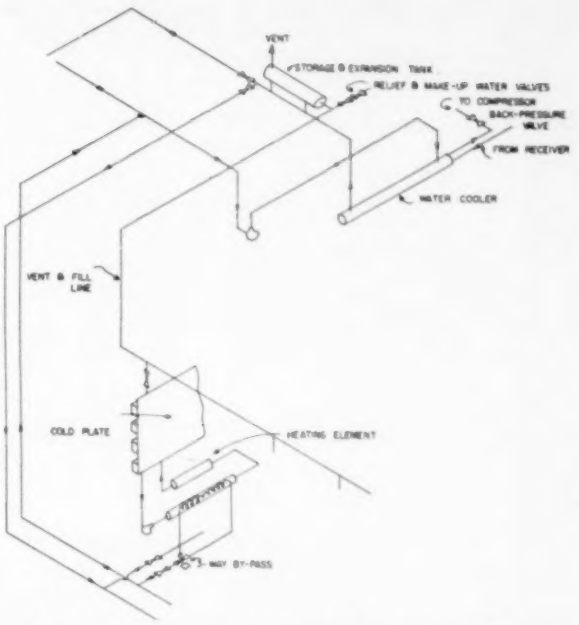

FIG. 2. Diagrammatic representation of the cooling system.

EXPERIMENTAL RESULTS.—*Uniformity of temperature control.*—To check the uniformity of temperature control within a dew chamber, particularly in the foliarsphere, the recording points of a 12-point thermocouple-type recording potentiometer were installed along the vertical axis in the center of a dew chamber that was set to maintain a 75°F air temperature with a temperature differential greater than 16°. The chamber used for the temperature check was loaded with test plants. One recording point was immersed in the water bath, and the other 11 were evenly spaced up to the top of the chamber from a point just above the water bath surface. The following temperature averages were noted over a 10-hour period: 95.7°F in the water bath, 77.2°F just above the water bath, 76.1°F at the level of the platform on which the pots were placed, and 75.5°F at the recording point in the foliarsphere; and from this point the temperatures dropped gradually to 74.0°F midway in the chamber, and rose to 75.4°F at the top of the chamber. These temperatures are listed in Table 1 as averages obtained from data recorded every hour during a 10-hour period. There was very little tempera-

TABLE 1.—*Temperature changes at various locations in a dew chamber adjusted for a 75°F air temperature with water-bath temperature about 95°F*

Interval (hours)	Chamber top	Chamber middle	Plant level (mid-leaf)	Base of pot	Just above bath	In bath
	°F	°F	°F	°F	°F	°F
1	74.3	72.5	72.9	72.9	74.7	94.1
2	76.5	75.2	77.0	77.0	79.2	97.6
3	75.2	73.8	74.3	74.7	77.0	93.2
4	76.1	74.3	75.6	77.9	78.3	97.6
5	75.6	74.7	77.0	77.4	78.3	96.4
6	74.3	72.9	73.4	74.3	74.7	92.7
7	76.5	75.2	77.4	78.3	79.2	98.6
8	75.2	74.3	76.5	77.0	78.8	95.5
9	73.4	72.0	72.9	74.3	74.3	92.7
10	76.5	75.2	77.4	77.9	79.2	98.6
Average	75.4	74.0	75.5	76.1	77.2	95.7

ture variation around the desired control point, even though the water bath was about 20°F above the controlled air temperature. If control was so good with a large differential between air and water bath, then better control could be expected with the lower differential under which these tests were made.

Amount of dew.—A thorough study was made at various temperatures to ascertain the proper differential to use for most consistent infection. In general, a differential of 8°F gave the most uniform dew deposition without run-off.

Critical temperatures for infection during dew.—Maximum pustule counts for 4 tests with each of 7 races of *P. graminis tritici* (11, 15B, 17, 38, 49, 56, and 139) occurred at an average temperature of 75°F. All of the maximum pustule counts occurred at 70–77°F. Analysis of variance of all tests yielded highly significant differences in pustule counts between temperatures of any one test. Very little infection occurred at 85° or at 60°F. Fig. 3 summarizes data from all of the temperature test runs. Infection at

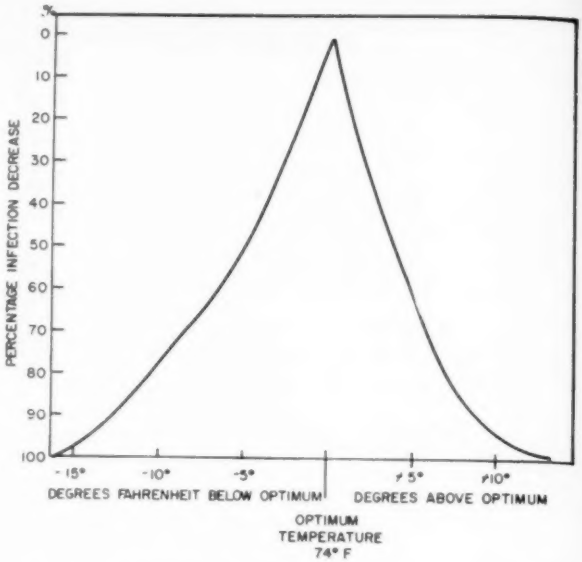

FIG. 3. Summation of infection data from 28 tests (4 tests with each of 7 rust races) at continuous temperature during 20 hours of dew.

incubation temperatures below the optimum decreases about 10% for each degree F. Slightly larger decreases in pustule numbers were encountered for every degree above the optimum.

DISCUSSION.—The precision of temperature control and dew control attainable with these dew chambers opens new horizons for investigation of critical conditions for infection, not only with the rust fungi but with other organisms as well. In this study, only continuous temperatures were investigated, but the equipment provides a means for determining the influence on infection of fluctuations in temperature during dew.

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RELATIONS BETWEEN ROOT KNOT AND FUSARIUM VASCULAR DISCOLORATION IN COTTON VARIETIES[1]

John P. Hollis [2, 3]

SUMMARY

The root-knot–fusarium-wilt disease complex was studied in 7 field sites in 4 cotton-growing regions of Louisiana. Experimental constants included soil fumigation for nematode control and 4 cotton varieties. Results common to all sites were: a) occurrence of *Meloidogyne incognita*, b) correlation of root knot and vascular discoloration, each rating individually in descending order in DPL 15 > Coker 100 Wilt > Plains = Auburn 56, c) rating of root-knot–vascular-discoloration ratios in descending order in DPL 15 > Plains = Auburn 56 > Coker 100 Wilt. These data enable separate ratings of varietal reactions to root knot and vascular discoloration and a statement of the problem of racial determinations within root-knot nematode species.

Analytical greenhouse studies (6, 7, 14) have somewhat clarified the relations among root-knot nematodes (*Meloidogyne* spp.), the wilt fungus (*Fusarium oxysporum* Schlecht. f. *vasinfectum* (Atk.) Snyd. & Hans.), and the root-knot–wilt-disease complex in cotton. Martin (6) isolated *Meloidogyne incognita incognita* (Kofoid & White, 1919) Chitwood, 1949, and *M. incognita* (Kofoid and White, 1919) subspecies *acrita* Chitwood, 1949, from a variety of hosts, and demonstrated a number of parasitic races of both subspecies on DPL 15 in the greenhouse. The nature of the races was not elucidated, but they probably are host specific (13). Wiles (14) tested several varieties of cotton, including Coker 100 Wilt and Auburn 56, for their reactions to 3 species and 2 subspecies of root-knot nematodes, and found them susceptible only to *M. incognita incognita* and *M. incognita acrita*. Martin, Newsom, and Jones (7) tested pure populations of several genera of plant parasitic nematodes commonly associated with cotton, both alone and in combination with *F. oxysporum* f. *vasinfectum*. Only *M. incognita incognita* and *M. incognita acrita* significantly increased the incidence of fungus wilt in 2 cotton varieties. The root-knot nematode isolates tested differed in ability to increase wilt incidence in Coker 100 Wilt.

This investigation tested the relevance of those greenhouse findings to field conditions in 4 of the 5 cotton-growing regions of Louisiana. Experimental constants or controlled variables were cotton-growing regions, varieties, and nematode control treatments. Measurable variables were root knot and vascular discoloration.

Unpublished evidence (personal communication, J. N. Sasser) on the morphology and host relations of *M. incognita incognita* and *M. incognita acrita* indicate that these organisms should, for the present and possibly in the future, be considered a single highly variable species or species complex. They are referred to in this paper as *Meloidogyne incognita* Kofoid & White, 1919.

Evidence is presented for: a) prevalence of *M. incognita* in the several cotton-growing regions, b) the absence of other root-knot nematode species, c) a preliminary evaluation of the problem of races of *M. incognita* under Louisiana conditions, d) a quantitative evaluation of the relation between root knot and vascular discoloration in the different cotton varieties, and e) the utility of the Horsfall-Barratt grading system (3) for root-knot evaluation in the field.

EXPERIMENTAL METHODS.—*Design of experiments.*—The experiments, at 7 locations, were a split-plot design within randomized blocks. The first 3 experiments were on a large scale, with 4-row variety plots 400–1200 feet long randomized within 16-row treatment plots, each containing 128 rows. The last 4, of compact design, with 4-row plots 50 feet long combining the various treatments and varieties, were 16 rows 400 feet long or 32 rows 200 feet long, depending on block arrangement.

Site selection, preparation, and soil fumigation.—Sites were selected for moderate to severe root-knot–wilt infestations under continuous cotton culture. Experiments 1, 2, and 3 were near Shreveport, on brownish-red Red River bottom soils; 4 and 5 were near Washington, in the upper rice-growing region, on Mississippi terrace soils; 6 was near Minden, on brownish-gray interior coastal-plains soil; and 7 was near St. Joseph, on gray Mississippi river bottom soil. All but site 6 were sampled for nematodes before and after soil fumigation. Larvae of *Meloidogyne* were found only in sites 1, 4, and 7, although root knot was later abundant in all sites, which is typical of fallow fields during winter or between crops in Louisiana. Species of *Tylenchorhynchus*, *Helicotylenchus*, and *Trichodorus* were found, respectively, in 6, 4, and 5 of the sites. Nematodes of these genera, alone and in combination with *F. oxysporum* f. sp. *vasinfectum*, have been found nonpathogenic to cotton

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in greenhouse studies (7). *Hoplolaimus tylenchiformis* Daday, 1905 (Syn. *H. coronatus* Cobb, 1923), found pathogenic to cotton under selected conditions by Krusberg and Sasser (5) in field and greenhouse tests, was present in 3 sites. *Xiphinema americanum* Cobb, 1913, *Criconemoides* spp., and *Pratylenchus* spp. were found, respectively, in 2, 2, and 3 of the sites. Apart from the report of Smith (9) that *Pratylenchus pratensis* (probably *P. brachyurus* (Godfrey, 1929) Goodey, 1951) may be implicated in fusarium wilt, nothing is known of the relations of these nematodes to cotton.

During the period of soil preparation and fumigation, soil moistures were high and soil temperatures were 45–55°F. In most cases a winter cover crop was plowed under before ridging for row spacing. Soil fumigant[4] (2 gal./A.) was applied 2–3 weeks before planting in the row with a tractor-drawn pressurized-flow applicator—in the furrows in some experiments; in the ridges in others. Depth after leveling the ridges was 6 in.

Grading of results and analysis of data.—Twenty plants, dug at random from the border rows of each plot, were individually scored for root knot and vascular discoloration. In the 7 experiments 4480 plants were scored—4–6 weeks before harvest except in experiments 4 and 6, which were graded at harvest. Root knot was graded on the scale of Horsfall and Barratt (3). Roots and stems were then cut longitudinally and rated for vascular discoloration: 0, none; 1, slight; 2, moderate; 3, severe. Yields of seed cotton were taken in 4 experiments.

Supplementary wilt and additional vascular discoloration readings could not be taken because the smallness of the plots in 4 of the experiments prohibited the removal of plants for coincident root-knot determinations. Further, vascular discoloration ratings are always superior to wilt determinations for correlations with root knot, because of the close association of *F. oxysporum* f. *vasinfectum* and *M. incognita* in the root system of the plant. Still, wilt and total plant reactions are genuinely correlated with yield and should be recorded whenever possible, particularly in plant-breeding work.

The vascular discoloration encountered, typical of that caused by *F. oxysporum* f. *vasinfectum*, was correlated with plants that wilted during a brief drouth in midsummer.

Analyses of variance were made of the root-knot, vascular-discoloration, and yield data in all experiments. Analyses were also made of the regression of root knot on vascular discoloration in experiment 1 and on each set of data in Tables 1, 2, and 3. Average Horsfall-Barratt grades from each plot were used directly for statistical analysis of all root-knot data. Vascular discoloration ratings were totals from each plot. The highest possible plot total would be 20 plants × grade 3, or 60. Totals were adjusted to percentages of 60 with the factor 1.67. For statistical analysis, the percentages were converted to angles from the table in Snedecor (11).

[4] Dowfume W-85—83% 1,2-dibromoethane and 17% solvent—furnished for some of the experimental applications by the Dow Chemical Company, Greenville, Mississippi.

TABLE 1.—*Root knot caused by Meloidogyne incognita in 4 cotton varieties at 7 locations in Louisiana in 1957*

Experiment	Mean root-knot grades[a]				LSD 05
	DPL 15	100 Wilt Coker	Plains	Auburn 56	
1[c]	3.54	2.44	1.04	1.11	0.64
2	3.26	2.11	1.28	0.95	0.62
3	0.86	0.37	0.35	0.42[b]	0.39
4	2.53	2.00	1.13	1.14	n.s.[d]
5	0.27	0.12	0.04	0.04	n.s.
6	4.29[b]	3.79	3.16	2.43	n.s.
7	2.75	2.03	0.92	0.86	0.71
Mean	2.50	1.84	1.13	0.98	

LSD at 5%, 0.52; at 1%, 0.72

[a] The Horsfall-Barratt system (3) comprises 12 grades based on percentage ranges equally easy to distinguish. Twenty plants from each plot were graded individually for per cent of root system knotted. Grades 1, 2, and 3 correspond, respectively, to average percentages of 2.0, 6.0, and 12.0.

[b] Plants were dead and lateral roots decomposed in DPL 15 in experiment 6, and Empire was substituted for Auburn 56 in experiment 3. Prior to statistical analysis of the tabular data, these values were estimated by the iterative method of Snedecor (11).

[c] Differences between experiments were highly significant although means and LSD are not shown.

[d] n.s. means nonsignificant.

Identification of root-knot nematodes.—During the grading operations, root and soil samples were collected from each cotton variety in the sites. Root samples were preserved in a mixture of 5% formaldehyde and 1% glacial acetic acid. Mixtures of roots and soil, identified by site and cotton variety, were added to clay pots in the greenhouse and planted to Rutger's tomato (*Lycospersicum esculentum* Mill.). Root samples from these plants were preserved and used for root-knot-nematode species identification by comparing perineal patterns of the adult females with those published by Chitwood (1). The soil cultures were then replanted with Spanish peanut (*Arachis hypogaea* L.) to test for the possible presence of *M. hapla* Chitwood, 1949, and *M. arenaria* (Neal, 1889) Chitwood, 1949, according to the practice of Sasser (8).

EXPERIMENTAL RESULTS.—Excessive rainfall around planting and harvest is reflected in an absence of statistical significance in yield data on experimental sites where severe yield reductions had been reported in previous years. Yield data were taken in experiments 1, 2, 3, and 6 and were nonsignificant with regard to treatment and significant due to variety only in experiment 6. This influence extended to treatment and varietal differences in root knot, although the latter were significantly differentiated in 4 of the 7 experiments. Differences in root knot due to treatment were significant only in experiment 1. Treatment × variety interactions occurred in only 2 of

the 7 experiments, reflecting no significant trends in the data. Differences in vascular discoloration due to treatment were significant only in experiment 4. Varietal differences in vascular discoloration were highly significant in all experiments. The combined data on varietal reactions are the most valuable aspect of the results.

Mean root-knot grades from replicated plots of the 4 varieties in all experiments are in Table 1. Root-knot levels were different in the experiments, and highly significant between experiments. Ratings by experiment were: 6—severe; 1, 2, 4, 7—moderate; 3, 5—slight. Where the rating was "severe" the grower both fumigates and grows a resistant variety for economic yields. Lack of significance in the data from experiments 4 and 6 is directly attributable to a delay in the grading work until the harvesting period. Death and root decomposition of many plants of the highly susceptible DPL 15 variety depressed root-knot values. This effect was most pronounced in experiment 6. Lack of significance for varietal differences in experiment 5 was due to the extremely low level of root knot.

The relation of varieties, one to another, was consistent between experiments. DPL 15 rated highly susceptible; Coker 100 Wilt, susceptible; Plains and Auburn 56, moderately resistant. Differences in varietal reaction were highly significant.

Vascular discoloration (Table 2) caused by *F. oxysporum* f. *vasinfectum* was much less than expected in Coker 100 Wilt, differing significantly from the values for Plains and Auburn 56. Since the varieties exhibited similar relative reactions to vascular discoloration and root knot, it might be assumed that they possess equivalent resistance or susceptibility to both elements. This assumption is invalid because vascular discoloration is a dependable variable, regulated to a large extent by the different levels of root knot among the varieties. An analysis of the regression of root knot on vascular discoloration in the data of experiment 1 was highly significant and characterized by the equation

$$Y = 8.91X + 1.03$$

Root-knot–vascular-discoloration ratios are in Table 3. The varieties retained their identity in all experiments, as shown by the significant differences between means. Coker 100 Wilt exhibited less vascular discoloration per unit of root knot than did the other varieties. Although susceptible to root knot, it should be more resistant than Plains and Auburn 56 to vascular discoloration in the absence of root-knot nematodes under prevailing Louisiana conditions.

Separate ratings for susceptibility of the varieties to root knot and vascular discoloration are in Table 4. The values are percentages relative to the reactions of DPL 15. The superiority of Plains and Auburn 56 on nematode- and wilt-infested soil in Louisiana is primarily due to their root-knot resistance. Jones (4) presented interesting data on this point. Among 8 outstanding varieties of cotton tested in the Red River Valley over a 3-year period, the average percentages

TABLE 2.—*Vascular discoloration caused by Fusarium oxysporum f. vasinfectum in 4 cotton varieties tested at 7 locations in Louisiana in 1957*

	Mean vascular discoloration[a]				
Experiment	DPL 15	Coker 100 Wilt	Plains	Auburn 56	LSD 05
1[c]	39.65	17.27	9.93	9.64	8.21
2	40.75	6.57	6.31	1.18	6.56
3	17.94	1.63	4.41	7.08[b]	5.93
4	35.54	19.50	11.14	12.49	8.51
5	47.42	16.11	6.86	15.89	9.92
6	90.00	31.15	31.00	26.17	13.93
7	64.99	24.47	12.93	14.73	18.19
Mean	47.66	16.67	11.80	12.06	

LSD at 5%, 9.87; at 1%, 13.56.

[a] The plants graded for root knot were also graded for vascular discoloration after cutting roots and stems longitudinally: 0 = none, 1 = slight, 2 = moderate, 3 = severe. The grades were totaled for 20 plants per plot, adjusted to percentages with the factor 1.67, and converted to angles for statistical analysis from the table of Bliss in Snedecor (11).

[b] Empire variety was substituted for Auburn 56 in this experiment. Prior to statistical analysis of the tabular data, the value for Auburn 56 was calculated with a formula listed by Snedecor (11).

[c] Differences between experiments were highly significant, although means and LSD are not shown.

TABLE 3.—*Vascular discoloration in terms of root knot (V. D./R. K.) in 4 cotton varieties at 7 locations in Louisiana in 1957*

	Vascular discoloration/Root knot			
Experiment	DPL 15	Coker 100 Wilt	Plains	Auburn 56
1[a]	11.20	7.08	9.55	8.68
2	12.50	3.11	4.93	1.24
3	20.86	4.41	12.60	16.86
4	14.05	9.75	9.86	9.86
5	175.62	134.25	171.50	171.00
6	20.98	8.22	9.81	10.77
7	23.63	12.05	14.05	17.13
Mean	39.83	22.55	33.19	33.65

LSD at 5%, 8.18.

[a] Differences between experiments were highly significant, although means and LSD are not shown.

TABLE 4.—*Relative susceptibility of cotton varieties to root knot, vascular discoloration, and root-knot–vascular-discoloration complex in field experiments at 7 locations in Louisiana in 1957*

	Per cent of relative susceptibility[a]		
Variety	Root knot	Vascular discoloration	Complex
DPL 15	100	100	100
Coker 100 Wilt	74	65	48
Plains	45	83	37
Auburn 56	39	85	33

[a] Root knot grades and vascular discoloration angles, the latter in terms of root knot (V. D./R. K.), were converted to percentages relative to DPL 15 = 100%. The disease complex is a product of the percentage of root knot and vascular discoloration.

of increase in yield with fumigation for nematode control were, by variety: Coker 100 Wilt, 73.4; Plains, 34.0; and Auburn 56, 25.2. The response of these wilt-resistant varieties on nematode- and wilt-infested sites to soil fumigation for nematode control is undoubtedly related to their relative susceptibility to root-knot nematodes.

All sites were infested with *Meloidogyne incognita*. Reactions of Rutger's variety of tomato to the different isolates showed that, in general, root-knot inocula from different cotton varieties were equivalent within each site. Observed differences in severity were correlated only with site. Relative severity ratings on tomato were: site 1, severe; 2, slight; 3, data not recorded; 4, moderate; 5, severe; 6, very slight; 7, very severe. The most severe root knot on cotton, in experiment 6, was least severe on tomato, the symptoms resembling those caused by *M. hapla*.

Discussion.—Smith (10) summarized the relative ratings of leading cotton varieties in Alabama in 1953 with regard to fusarium wilt and root-knot resistance. Per cent wilt was 9.6 for the Cook 307 derivatives, including Auburn 56; and 31.7 for the Clevewilt derivatives, including both Coker 100 Wilt and Plains. The root-knot index was 73.4 for the Cook 307 group, and 87.2 for the Clevewilt group. The present data emphasize the marked differences in reactions of the tested varieties to root knot and wilt (determined as vascular discoloration) in Louisiana as compared with Alabama. On a relative basis, Fusarium resistance of Auburn 56 has been broken to the approximate level of Plains, that of Plains has been maintained, and that of Coker 100 Wilt has been enhanced. Root-knot resistance in Auburn 56 has dropped also to the approximate level of Plains, although both remain relatively high. Root-knot resistance of Coker 100 Wilt has broken markedly. The prevalent nematode species causes severe root knot in this variety, but it is relatively inefficient in intensifying vascular discoloration.

Under Louisiana conditions Auburn 56 is most resistant to root knot, and Coker 100 Wilt is most resistant to vascular discoloration. Varietal and treatment data on the root-knot–fusarium-wilt complex were equivalent in several crop regions of Louisiana. Practical recommendations from results in one region should therefore be generally applicable to the other cotton-growing regions.

The results of these field experiments cast some light on variability of *M. incognita* (13). Relational bases of variation were formulated by Waggoner and Wallin (13) for *Phytophthora infestans*, the causal agent of potato and tomato late blight, and definitions were given for pathic, hostic, environmental, and developmental races. Populations of *M. incognita* differed significantly in the average quantity of disease they produced on 4 cotton varieties in 4 crops regions of Louisiana. Before they can be designated pathic races in cotton, it will be necessary to ascertain whether they differ significantly under all environments and after all times of development encountered in field experiments. It will be of special interest to test for possible interactions between indigenous nematode populations and soil types. Such interactions would indicate the existence of edaphic races.

Extension of the host range of the root-knot-nematode isolates from cotton to tomato resulted in differential reactions indicative of hostic races (6). They may be provisionally called hostic races subject to appropriate reservations. Difficulties may be anticipated in the separation of host and development effects. Root-knot development is a geometric function of time, which appears to exert a $(x)^3$-fold effect on disease expression. The problem is complicated also by host differences in the attraction of larvae and their penetration of roots (8).

The discovery of "races" of the root-knot nematode by Christie (2), later separated as morphological species by Chitwood (1), foreshadowed the recognition and analysis of morphologically indistinguishable variants, separated only by host effects (6). Similarly, the occurrence of environmental races may be presaged by the report of Thomason (12) that species of *Meloidogyne* differed in their responses to temperature.

Preliminary evidence suggests the existence of hostic, and the probable existence of pathic, races of *M. incognita*. Two important rules should be observed for the racial characterization of a root-knot nematode species. The types and numbers of hosts tested should be selected carefully and specified exactly because their selection will prejudice the results to a large degree. In order to confine a study to naturally occurring variants, as many as possible of the relational bases of variation should be included in initial experiments. This can be best accomplished by beginning such studies in the field.

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A NEW VIRAL DISEASE OF TOMATO AND PEPPER[1]

P. M. Miller[2] and H. H. Thornberry

SUMMARY

An apparently new virus was found in tomato in 1952 and in pepper in 1953. The virus, tomato atypical mosaic virus (TAMV), is most probably a strain of tobacco mosaic virus (TMV). TAMV is similar to TMV in size, shape, serological reactions, infectious dilution end-point in expressed juice, and capacity to infect certain hosts. TAMV, however, is inactivated in 10 minutes at 72°C, is unstable to freezing and thawing, does not infect certain varieties of beans susceptible to TMV, does not cross-protect against TMV in tomatoes, and under field conditions induces symptoms in tomato and pepper unlike those from TMV. TAMV seemingly is of potential importance.

A new disease of field tomato plants, *Lycopersicum esculentum* Mill., was found near Ridgefarm, Illinois, in 1952, by M. B. Linn. Although the symptoms suggested a mineral deficiency and were unlike any known viral disease, a preponderance of affected plants adjacent to mixed vegetation along a fence aroused suspicion that a virus was involved. Preliminary diagnostic investigations on crude extracts from the foliage of affected plants revealed an infectious agent that seemed similar to TMV (tobacco mosaic virus). The extract contained numerous rods of about 15×300 mμ, and the highly infectious agent induced primary necrotic local lesions on *Nicotiana glutinosa* L. and systemic mottling on Turkish tobacco, *N. tabacum* L. This virus from tomato is herein designated as TAMV (tomato atypical mosaic virus). In 1953 the disease was observed in other areas of the state. An undescribed disease of pepper plants, *Capsicum annuum* L., also was found in a field near Chicago. Expressed sap from triturated leaves of affected pepper plants likewise contained an infectious agent presumably the same as that from TAMV-diseased tomatoes since the agents were indistinguishable in preliminary diagnoses. Further investigations provided additional results, published in part as an abstract by Miller (13), and reported here in detail.

DESCRIPTION OF DISEASES.—The diseases are herein designated and described as tomato atypical mosaic and pepper atypical mosaic although the causal virus induces symptoms that are typical of infections by tobacco mosaic virus in some other plants. The word "mosaic" suggests a relationship to the common mosaic viral diseases of these plants; the word "atypical" serves 2 purposes: it denotes individual entities and suggests that the new entities are related to typical mosaic diseases.

Tomato atypical mosaic.—Under field conditions, tomato atypical mosaic primarily affects the foliage. The leaves of affected plants develop interveinal chlorosis, with the veins remaining green. The areas deficient in chlorophyll may in time become nearly white, and in some cases develop necrosis that is light brown to dark brown or black. The chlorotic areas occur most frequently on leaflets located midway between the apex and base of the plant, and appear more like symptoms of mineral imbalance than of viral infections. Distinct foliar chlorosis also occurs on the lower portions of affected plants. Faint foliar chlorosis, without necrosis, develops on the younger portions of the plants, but not on very young leaves. Foliar symptoms slightly resemble those of aucuba mosaic. Foliar mottling is not associated with field infections known to be caused by TAMV. Stems, fruits, blossoms, or roots reveal no observable symptoms. General plant vigor is reduced.

Pepper atypical mosaic.—Under field conditions, pepper atypical mosaic is difficult to recognize with assurance. The leaves are pale green, with only a slight interveinal chlorosis and without necrosis. The fruits appear to be incompletely expanded, but without other noticeable characteristic. The stems, blossoms, and roots of diseased plants have no observable symptom. The general vigor of diseased plants is slightly reduced. These symptoms, obviously, could

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The studies, in part, were conducted by the senior author in connection with a graduate research course in plant pathology. The authors are grateful to A. E. Vatter and D. D. Hickman for the electron microscopy.
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be easily confused with usual variations in the growth of uninfected plants or plants with degrees of mineral imbalance.

Symptoms on greenhouse-grown tomatoes, peppers, and some other susceptible plants are described in the section on experiments and results.

DISTRIBUTION OF THE DISEASES.—The distribution of the diseases on field tomatoes and on field peppers suggests that the causal virus may be widespread. After the disease of tomatoes was recognized, in 1952, it was observed in 1953 on tomatoes in 2 additional areas in Illinois: Danville and Sterling, about 100 miles apart. In 1954, it was found on peppers in vegetable-growing areas near Chicago, Sterling, and Danville. A similar disease of tomatoes occurred in Indiana in 1952, according to R. W. Samson (unpublished), who expressed an opinion that the disease was caused by the "burning strain" of TMV.

MATERIALS AND METHODS.—Three isolates of viruses were used in the studies: 1) a local-lesion isolate of TAMV obtained from *Nicotiana glutinosa* L. plants previously inoculated with expressed juice from a field tomato plant showing symptoms of tomato atypical mosaic disease; 2) a mass isolate of the pepper atypical mosaic virus (PAMV) derived from a systemic infection of Turkish tobacco, *N. tabacum* L., previously inoculated with expressed juice from a field pepper plant showing symptoms of the pepper atypical mosaic disease; 3) a mass isolate of TMV, *Marmor tabaci* var. *vulgare* Holmes, Johnson's Strain No. 1, secured from W. C. Price in 1938 and maintained in living Turkish tobacco plants. The supply of the 3 viruses used in the studies was obtained from a comparable source, infected Turkish tobacco plants.

Inoculum used in the studies on host range and host reactions consisted of aqueous dilutions of juice expressed from the diseased tobacco plants. The infected tissue was triturated in a hand-operated food chopper, and the liquid was separated from the pulp by wringing by hand through 4-ply cheesecloth. The liquid was further clarified by centrifuging 15 minutes at 500 G.

Partially purified preparations of the 3 viral isolates were obtained by 2 differential centrifugations: 30 minutes at 5,000 G and 90 minutes at 30,000 G.

Antiserum to TAMV or to TMV was prepared by 5 intraperitoneal injections of partially purified preparations of the viruses into either guinea pigs or chickens. Antiserum from chickens was extracted with ethylene dichloride to remove fatty substances.

Bioassays for the activity of TAMV or TMV, unless otherwise stated, were on leaves of *Nicotiana glutinosa* L., and were done with viral suspensions in 0.1*M* phosphate buffer at pH 7.0 containing 7% abrasive, 800-mesh aluminum oxide powder. Inoculations consisted of stroking the upper surface of leaves with a cloth pad moistened with the viral suspension. Stroked leaves were rinsed with tap water soon after applying inoculum. In estimating the amount of active virus from the number of local lesions, it was assumed that the dispersion of infective particles was constant.

TABLE 1.—*Comparative dilution [a] end-point of tomato atypical mosaic virus (TAMV) and tobacco mosaic virus (TMV) in crude extracts from infected Turkish tobacco plants*

Dilution	Number of lesions [b]	
	TAMV	TMV
10^{-3}	54	49
10^{-4}	28	30
10^{-5}	8	8
10^{-6}	0.7	0.5
10^{-7}	0.3	0.5
10^{-8}	0	0

[a] The extracts were diluted in 0.1*M* phosphate buffer at pH 7.0.

[b] Mean number of lesions per half-leaf from a total of 16 half-leaves in 2 experiments.

EXPERIMENTS AND RESULTS.—A preliminary comparison of the infections from TAMV and PAMV on some common hosts strongly indicated that the 2 viral isolates were indistinguishable. PAMV was not investigated further.

Morphology of the TAMV was determined with an EMB electron microscope from mounts of partially purified preparations shadowed with palladium. The mounts contained numerous rods, the greater portion of which were about 15×300 mμ.

The infective dilution end-point of expressed juice containing TAMV or TMV was determined by infectivity tests on *N. glutinosa* from dilutions in 10-fold series up to 10^{-8}. Each extract was infectious at 10^{-7} but not at 10^{-8} (Table 1).

Thermal inactivation of TAMV was determined on samples of infectious crude juice diluted with distilled water to 10^{-2}. Samples of TMV in crude extract served as controls. Two ml of the diluted juice in Pyrex culture tubes, 16×150 mm and 1 mm thick, were brought 10°C below the desired temperature by heating the tubes during vigorous agitation over a small gas flame. The tubes were then heated in a water bath at the desired temperature ±1°C. After heating for a standard period, 10 minutes, the tubes

TABLE 2.—*Comparative stability of tomato atypical mosaic virus (TAMV) and tobacco mosaic virus (TMV) in crude extracts at a dilution of 10^{-2} in 0.1M phosphate buffer to the contentration of H ions for 10 minutes at 21°C*

pH	Number of lesions [a]	
	TAMV	TMV
2.0	22	15
2.5	22	17
3.0	31	29
7.0	58	77
9.0	40	58
9.5	129	112
10.5	27	28
10.5	5	3
11.0	0	0

[a] Mean number of lesions per half-leaf on a total of 30 half-leaves in 3 experiments.

were removed from the bath and quickly cooled in tap water. The samples were diluted 10-fold in 0.1*M* phosphate buffer at pH 7.0 for viral assay on *N. glutinosa*. The results show that 10 minutes at 72°C completely inactivated TAMV, whereas TMV under similar conditions required 10 minutes at 85°C for complete inactivation. There was no inactivation of TAMV at 60°C or below, but 76% and 97% inactivation, respectively, occurred in 10 minutes at 65°C and 70°C. The results for TMV are from 1 experiment on a total of 6 leaves of *N. glutinosa;* those for TAMV are from 2 experiments on a total of 12 leaves.

Stability of TAMV to pH was determined by adjusting 10^{-2} dilutions of crude infectious juice to the desired pH with 0.1*N* NaOH or HCl and allowing the solutions to remain at the adjusted pH for 10 minutes at room temperature, about 21°C. At the end of the 10-minute treatment, the liquids were quickly cooled and diluted 10-fold in 0.1*M* buffer at pH 7.0, and later adjusted to pH 7.0 for assay of active virus. As controls, samples of crude juice containing TMV were treated similarly. The results (Table 2) reveal that TAMV and TMV responded similarly, i.e. inactivation was complete at pH 11.0, marked at pH 10.5, and slight at pH 10.0. The observed reduction of activity at pH 3.0, 2.5, and 2.0 may not have resulted from inactivation but from an aggregation of infectious viral particles that were not subsequently dispersed by dilution at pH 7.0.

The stability of TAMV to freezing at —20°C was determined on samples of infected tissues, of crude extract from unfrozen tissues, and of dilutions of this crude extract. Survival of TAMV was 35% if frozen in tissues, and 24% if frozen either in crude juice or in dilutions of the crude juice. Survival of TMV was 90% if frozen in diluted crude extract; survival under other conditions was not determined. The infectivity of the samples was assayed at a dilution of 10^{-3} with corresponding control samples on opposite halves of the leaves. The controls for TAMV and their respective infectivities were: tissues, 91 local lesions per ½ leaf from 10 leaves in 1 test; crude extract, 51 local lesions per ½ leaf from 10 leaves in 1 test; and diluted crude extract, 57 local lesions per ½ leaf from 26 leaves in 3 tests. The infectivity of the control for TMV was 141 local lesions per ½ leaf from 26 leaves in 3 tests.

Stability of TAMV and TMV during storage at 4°C was determined on samples of a 10^{-2} aqueous dilution of crude juice. Samples of TAMV stored 0, 4, 10, and 20 days and then assayed on *N. glutinosa* produced, respectively, 59, 20, 17, and 1.7 local lesions per leaf. TMV stored similar periods and assayed on *N. glutinosa* produced, respectively, 115, 106, 81, and 84 local lesions per leaf. Thus, TAMV is unstable at 4°C, whereas TMV is relatively stable.

Cross-protection in tomato, variety Blackhawk (Illinois 97A), by the TAMV virus against subsequent infections by TMV was determined by inoculating young tomato plants with TAMV 3 weeks before inoculating with TMV. Tests for TMV in the TAMV-infected tissues of tomatoes were made on leaves of Scotia beans, which are insusceptible to TAMV. Lack of suitable assay plants prevented a reciprocal cross-protection test. TAMV did not cross-protect against TMV under these conditions. Numerous necrotic local lesions were obtained.

TABLE 3.—*The effect of normal guinea-pig serum and guinea-pig antiserum for tomato atypical mosaic virus (TAMV) on the infectivity of partially purified preparations of TAMV and tobacco mosaic virus (TMV)*

Virus	Treatment	Number of lesions [a]	Infectivity as % of control
TAMV	None, control	86	100
TAMV	Normal serum	38	44
TAMV	TAMV antiserum	9	10
TMV	None, control	60	100
TMV	Normal serum	27	45
TMV	TAMV antiserum	13	22

[a] Mean number of lesions on a total of 10 leaves in 2 experiments.

Serological relationship between TAMV and TMV was determined by reactions from antiserum of TAMV and TMV. A precipitin reaction in capillary tubes was obtained at the junction of TAMV antigen and either TAMV or TMV antiserum, and also at the junction of TMV antigen and either TAMV or TMV antiserum. The TAMV antiserum strongly inhibited infections from either TAMV or TMV on *N. glutinosa* (Table 3). Since this inhibition was greater than the inhibition of infection by normal serum, the 2 viruses surely are serologically related.

Infectivity tests were made with TAMV and TMV on a number of plant species and commercial varieties. The tested plants were observed for reactions and assayed for detectable virus in tissues not previously receiving inoculum.

The results (Table 4) indicate that the reactions of the plants to infection by the 2 viruses were essentially similar. However, 5 varieties of beans were insusceptible to TAMV, but developed necrotic local lesions from TMV. One variety of cowpea, California Black Eye (Large), which is insusceptible to TMV, developed small reddish-brown necrotic local lesions on inoculated leaves in one test with TAMV, but not in later tests. Several species and varieties were without observable symptoms or detectable virus following the infectivity tests with either TAMV or TMV. Symptomless plants that contained assayable amounts of TAMV were *Antirrhinum majus, Physalis* sp. (ground cherry), *Solanum dulcamara,* and *Zinnia elegans.* These plants also were without symptoms following tests with TMV, but contained assayable amounts of TMV.

DISCUSSION AND CONCLUSION.—These diseases, though new to our knowledge of viral diseases of these crop plants, may have been present for some time and unrecognized because the commonly occur-

TABLE 4.—*Comparative reactions of species and varieties of plants to infectivity tests with tomato atypical mosaic virus (TAMV) and tobacco mosaic virus (TMV)*

Plant	Reactions [a]		Assay [a, b] of juice from plants tested with	
	TAMV	TMV	TAMV	TMV
Antirrhinum majus var. Tetraploid Giant ruffled mixture	None	None	+	+
Brassica rapa	None	None	—	—
Calendula officinalis var. Orange King Deep Orange Deep Crater	None	None	—	—
Callistephus chinensis var. Ball's Resistant Aster Royal Shell Pink	None	None	—	—
Capsicum annuum var.:				
Anaheim Chili	NLL	NLL	—	—
Banana	M, SNL	M, SNL	+	+
California Wonder	M, SNL	M, SNL	+	+
Cayenne Long Pod	NLL	NLL	+	+
Golden California Wonder	M, SNL	M, SNL	+	+
Harris Early Giant	M	M	+	+
Illinois F_3 1-1	NLL, M	NLL, M	+	+
Large Bell (mild)	NLL, M	NLL, M	+	+
Mango	NLL, SNL	NLL, SNL	—	—
Patrick Henry	NLL, M	NLL, M	+	+
Pimento Perfection	NLL, M, SNL	NLL, M, SNL	+	+
Red Chili	NLL, M	NLL, M	+	+
Ruby Giant	M	M	+	+
Ruby King	NLL, SNL	NLL, SNL	?	?
Sterling	NLL, M	NLL, M	+	+
World Beater	NLL, SNL	NLL, SNL	—	—
Cucumis sativus var. Straight Eight	None	None	—	—
Dahlia sp. var. Unwin's Dwarf Hybrid Mixed	None	None	—	—
Datura stramonium, Jimson weed	NLL	NLL	—	—
D. Wrightii, Angels Trumpet	NLL	NLL	+	+
Helianthus annuus var. Sungold Double Yellow Sunflower	None	None	—	—
Hibiscus esculentus var. Perkins Green Dwarf Long Pod	None	None	—	—
Lycopersicum esculentum var.:				
Bonny Best	M	M	+	+
Blackhawk (Illinois 97-A)	M	M	+	+
Garden State	M	M	+	+
Garden State, colchicine segregate	M	M	+	+
Garden State, tetraploid	M	M	+	+
Miscellaneous hairy variety	M	M	+	+
Pink Harry	M	M	+	+
Prichard	M	M	+	+

(*continued next page*)

ring symptoms are atypical of viral infections. The syndromes of the diseases, nevertheless, are distinguishable from those caused by the sap-transmissible viruses listed by Weiss and O'Brien (18). They also seem distinct from published descriptions of other sap-transmissible viral diseases, such as aster ringspot virosis of pepper (1), eggplant mosaic in pepper and tomato (6), pepper mosaic (6), pepper mottling mosaic (5), pepper or tomato mottling and calyx enlargement (11), pepper ring spot disease (2, 4), pepper veinbanding mosaic (16), pepper yellow-pod disease (2), pepper viral diseases caused by the latent strain or the South Carolina mild mottling strain of tobacco mosaic virus (12), tomato bronze leaf (6), tomato ring spot disease (15), and tomato yellow-net virus disease (17).

TAMV is unlike the 54 strains of TMV obtained by Jensen (8). Although the green mottling symptoms produced in Turkish tobacco by Jensen's 501 and 502 strains might be confused with symptoms arising from TAMV in this host, strains 501 and 502 are certainly not similar to TAMV, since they are not inactivated in 10 minutes even at 89°C, a much higher temperature than 72°C, which inactivates TAMV. Other strains of TMV mentioned by Holmes (7) seem distinct from TAMV on the basis of symptoms in Turkish tobacco and tomato plants.

The economic importance of the diseases is not assessable from present information. Although field-grown plants do not develop symptoms of mottling, there seemingly is a reduction in the vigor of plants and in the quality and quantity of fruit. The diseases have the potential to become economically important because of such factors as high titre of virus in dis-

TABLE 4.—(continued)

Plant	Reactions [a]		Assay [a, b] of juice from plants tested with	
	TAMV	TMV	TAMV	TMV
Mirabalis valana var. Marvel of Peru Mixture	None	None	–	–
Nicotiana glutinosa	NLL	NLL	–	–
N. repanda	NLL, M	NLL, M	+	+
N. tabacum, Turkish tobacco	M	M	+	+
Petunia hybrida var. Dwarf Hybrida Celestial Improved, Extra Dwarf Strain	None	None	–	–
Phaseolus vulgaris var.:				
Bountiful	None	None	–	–
Early Golden Cluster Wax	None	NLL	–	–
Great Northern	None	NLL	–	–
Idaho Refugee	None	NLL	–	–
Kentucky Wonder	None	None	–	–
Kentucky Wonder Wax	None	None	–	–
Pinto	None	NLL	–	–
Scotia (Striped Creaseback)	None	NLL	–	–
Tendergreen	None	None	–	–
Physalis sp., ground cherry	None	None	+	+
Pisum sativum var.:				
Brown Sugar Crowder	None	None	–	–
Thomas Laxton	None	None	–	–
Raphanus sativus var. White Icicle	None	None	–	–
Salvia splendens var. Earliest Extra Dwarf Red	None	None	–	–
Solanum carolinense, Horse nettle	M	M	+	+
S. dulcamara, Bitter nightshade	None	None	+	+
S. integrifolium, Ornamental eggplant	NLL	NLL	+	+
S. melongena, Eggplant Var.:				
Black Beauty	NLL	NLL	–	–
Florida High Bush	NLL	NLL	–	–
Improved New York	NLL	NLL	–	–
Vaughn's Hybrid	NLL	NLL	–	–
Spinacia oleracea, spinach	None	None	–	–
Tropaeolum majus, nasturtium var. Dwarf Double Golden Globe Golden Yellow	None	None	–	–
Vigna sinensis, cowpea var.:				
Calif. Black Eye	None	None	–	–
Calif. Black Eye Large	None [c]	None	–	–
Zinnia elegans var. dahlia-flowered Canary Bird Canary Yellow	None	None	+	+

[a] Legend for reactions and assays: NLL = necrotic local lesions; M = mottling; SNL = stem necrotic lesions; + = positive assay for virus in tested plant; – = negative assay for virus in tested plant; ? = questionable assay for virus in tested plant.

[b] Assays for the increase of virus in the tested plants were made with expressed juice from uninoculated portions of plants. Leaves of *Nicotiana glutinosa* or Anaheim chili pepper were used in the assays.

[c] Small reddish-brown necrotic lesions in one test, but not in subsequent tests.

eased plants, mechanical transmission, susceptibility of a wide range of hosts, and probably insect transmission.

The origin of TAMV is obviously unknown. Although the known characteristic of TAMV strongly suggests a relationship to TMV, TAMV has some properties suggesting a relationship to such viruses as cucumber virus 3 and 4 or tobacco aucuba mosaic virus. Knight (10) recommended that cucumber viruses 3 and 4 not be considered strains of TMV. Since the exchange of viral parts in mixed infections has been demonstrated with animal and bacterial viruses (3), one is prone to conjecture that TAMV may have originated by such a phenomenon involving TMV and possibly cucumber virus 3 or 4 in some common host or biovector. A more generally acceptable origin of TAMV is by mutation or by selection of an entity from a mixed population. There is no evidence to suggest that TAMV is a contaminated culture of TMV.

The data herein support the conclusion that TAMV is related to TMV, presumably a variety, although they are not yet sufficiently complete to comply with all 7 criteria for the relationship suggested by Knight (9). A proposal to classify TAMV as a variety of TMV and to assign an appropriate Latin trinomial to it is forthcoming in a paper based in part on additional characteristics of the virus (14).

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THE MECHANISMS OF FUSARIC ACID INJURY[1]

Ernst Gäumann[2]

SUMMARY

Fusaric acid molecules are carried within tomato shoots in the transpiration stream and concentrated particularly in the stem pith, the tissues above and between the vascular bundles, and certain interveinal areas of the leaves. Even within the first 48 hours about 10% of the amount taken up is decarboxylated by the host, and thereby possibly increased in toxicity; a further 8–24% (according to the tomato variety) is methylated, and thereby, for the most part, detoxicated.

Increasing concentrations of fusaric acid impair the water-permeability of the host protoplasts in different ways; thus, the harmful action at concentrations below $10^{-5}M$ is governed by the pyridine ring, and at concentrations above $10^{-4}M$ by the aliphatic side-chain in the β position. Hence, in *Rhoeo discolor* (L'Hér.), the sensitivity of the protoplasts to both these active groups is modified diversely by over- or under-nourishment with N, P, or K.

The semi-permeability (polar permeability) of the plasma boundary layers in tomato plants is injured by fusaric acid in such a way that metal ions, amino acids, and peptides escape from within the cell into the transpiration stream of the cell walls; hence, the physical pre-conditions for osmotic pressure—and, therefore, of turgor—are destroyed. In addition to this direct way, the turgor in the wilting portions of the shoot is reduced indirectly, by a cutting down of the water supply.

Chemically, fusaric acid molecules are little differentiated, and they therefore can insert themselves into the most varied functions and sub-structures of the host cells. Not only the molecule as a whole takes part in the injurious activity, but some of its chemical groups also interfere independently in the vital processes of the host. Thus, in *Rhoeo* protoplasts, the pyridine ring of fusaric acid in low concentrations impairs oxidative phosphorylation, while, in the same protoplasts, the aliphatic side-chain in the β position blocks the cytochrome oxidase in higher concentrations. Because of this multiplicity of action, the fusaric acid molecules are "veering in all directions," that is, they are pleiotropic. Merely because of their ability to chelate heavy-metal ions they can paralyze the most varied enzyme systems of a host. Their sites of activity within the host cells correspond in variety; thus, the receptors for impairment of semi-permeability occur mainly in the plasma boundary layers, whereas those for impairment of respiration are in the mitochondria.

Some harmful activities of fusaric acid, such as the chelation of heavy-metal ions, increase in intensity with rising concentrations (isodromic course of the curve of injury). Others, such as the impairment of the water-permeability of protoplasts, reverse their direction with rising concentrations; for example, from increasing water-permeability at lower toxin concentrations to "waterproofing" the protoplasts at higher toxin concentrations (enantiodromic course of the curve of injury). Because the distribution of fusaric acid in diseased plants shows a gradient from focal to distal parts and from "privileged" to "non-privileged" tissues, and, moreover, because it undergoes a permanent alteration during the course of the disease, the summation of the harmful activities of fusaric acid within a diseased plant is a highly complex resultant.

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Translated from the German by Dr. William B. Brierley, formerly at the University of Reading.

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In an earlier communication (12), the author attempted to describe our present knowledge of the chemistry of fusaric acid, the way in which it is formed, the range of its formation and activity, and some of the injury symptoms it evokes. In the present paper are considered some of the functional disturbances induced by fusaric acid that eventually lead to manifest disease of the host plant.

WHAT HAPPENS TO FUSARIC ACID IN THE HOST.—Fusaric acid molecules are small; their molecular weight is 179, as against 72,000 for diphtheria toxin A. Because of their small size and water-solubility they are extremely mobile; they are carried in the transpiration stream, travel with it (like mineral salts) in the micellar felting of the cell walls, and from the cell walls penetrate on all sides to the interior of the protoplasts. Chemically, their structure is simple and has little stability.

The distribution of fusaric acid inside tomato shoots.—If tomato cuttings are allowed to take up a definite amount of synthetic fusaric acid whose carboxyl group has been labelled with a C^{14}-atom, nearly one third of the given dose can be detected in the leaves by the end of the second hour (37). After 48 hours, radioactivity in the leaves is more than 10 times as high as in the stems, but manifest injuries, disproportionately severe, appear in the stems. The essential toxic processes that initiate pathological reactions in the plasma, finally leading to necrosis of the affected tissues, must therefore come into play very quickly, even at an early stage of incubation (36).

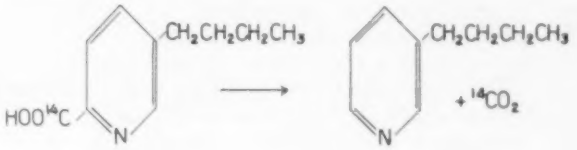
Fusaric acid is distributed as unevenly in stem tissues as in leaf tissues. In stems, radioactivity is found mostly in the tissues around and between the vascular bundles and in the pith. In leaves it is concentrated at certain points in the interveinal areas (which ultimately become necrotic) while the veins of the leaves, for example, become practically denuded of it. The causes of this predilection of fusaric acid for particular tissues of the host—or, alternatively, of the selective fixation of fusaric acid by these tissues—are still unknown.

Chemical changes in fusaric acid within the host.—If tomato cuttings are allowed to take up 150 mg of labeled fusaric acid per kg of fresh weight, then, according to the variety, only about 30–50% of the unmodified fusaric acid can be detected in them after 48 hours (38, 52); the remaining 50–70% has been transformed or split under the influence of the enzyme systems of the host. This chemical change of the fusaric acid within the host makes it hard to determine the amount of fusaric acid formed by the pathogens in question inside their host plants.

Up to the present, 2 changes that the fusaric acid molecules undergo inside the host plants have been established chemically: decarboxylation and methylation.

1) *Decarboxylation.*—Decarboxylation affects about 10% of the fusaric acid taken up by tomato cuttings; corresponding amounts of $C^{14}O_2$ can be detected in the atmosphere around them. This consumption of fusaric acid in the respiration of the host occurs independently of light and darkness and of whether the pH of the fusaric acid absorbed is 4.3, 6.0, or 7.2; hence, it is not conditioned by external factors (52).

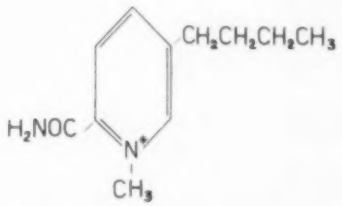
Provided the fusaric acid molecules undergo no change other than decarboxylation, the first cleavage product will be 3-*n*-butylpyridine, corresponding to the formula



Fusaric acid 3n-butyl-pyridine

In one sector of its activity—disturbance of the water-permeability of the protoplasts of the host cells, 3-*n*-butylpyridine is about 100 times as toxic as unmodified fusaric acid (Fig. 1C); in this respect the latter acts mainly as a *protoxin*, which increases its lethal toxicity by decarboxylation inside the host. Admittedly, 3-*n*-butylpyridine has not yet been demonstrated inside the host tissues; hence, we still do not know if the formula given here corresponds to biological reality.

2) *Methylation.*—Methylation of the N-atom in the pyridine ring, in conjunction with the formation of acid amide, leads by ways that are still unknown (perhaps by means of transmethylases of the host) to the quaternary ammonium-base of fusaric acid amide (38). This has the following structural formula



and is probably saturated by organic acid esters inside the protoplasts.

This quaternary ammonium-base of fusaric acid amide has lost the more important pathogenic characters of fusaric acid; thus, it no longer induces a wilt effect in tomato shoots, nor does it inhibit the germination of chlamydospores of *Ustilago zeae* (Beckm.) Ung. or the respiration of yeast cells in a Warburg apparatus; on the other hand, at lower concentrations its pyridine ring still causes excessive water-permeability of the protoplasts.

The methylation of the fusaric acid molecule at the N-atom of the pyridine ring thus leads to its far-reaching *detoxication*, therefore constituting an *anti-toxic defense reaction* of the tomato plant. This defense character also finds expression in the fact that

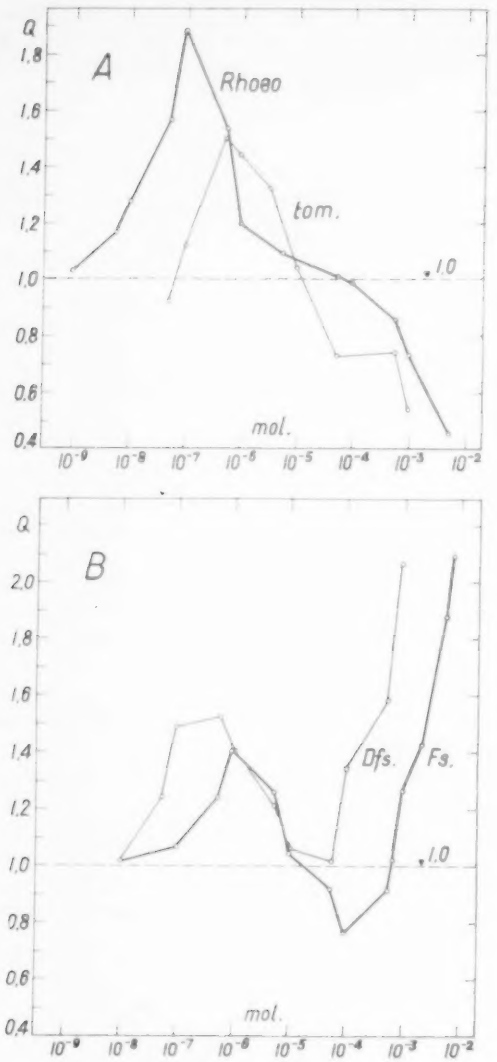


FIG. 1. The effect of increasing concentrations of fusaric acid and of certain other pyridine derivatives on the water-permeability of plant protoplasts. A) The effect of fusaric acid on the water-permeability of epidermal cells of *Rhoeo discolor* (curve *Rhoeo*) and medullary cells of young tomato shoots (curve *tom.*). B) The effect of fusaric acid (curve *Fs.*) and of dehydrofusaric acid (curve *Dfs.*) on the water-permeability of protoplasts of *Spirogyra nitida*. C) The effect of 3-*n*-butylpyridine on the water-permeability of protoplasts of *Rhoeo discolor* (curve *Rhoeo*) and of *Spirogyra nitida* (curve *Sp.*). *Abscissae:* molar concentration of solution. *Ordinates:* degree and type of injury to the water-permeability; quotients over 1 indicate a heightening, quotients below 1 a lowering, of water-permeability. (After Bachmann (1) and Gäumann and Loeffler (19)).

the relatively highly wilt-susceptible tomato varieties, Bonny Best and Tuckswood, deactivate, by methylation, only 8–9% of the fusaric acid administered, whereas, other things being equal, the highly resistant variety Red Currant deactivates 20–24%. Because of its pronounced capacity for methylation, the variety Red Currant is highly toxin-resistant to fusaric acid (38).

Similar methylations of pyridine and some of its derivatives at the N-atom of the pyridine ring are known in human medicine also; they transform the compounds in question into a state in which they can be eliminated from the animal or human body, thus becoming functionally inactivated (40, 61).

Methylation and decarboxylation of fusaric acid undoubtedly occur in different sites of activity of the cells; decarboxylation, for example, may perhaps take place at or in the mitochondria.

In their biological significance the 2 phenomena are antithetic. Decarboxylation, which affects about 10% of the absorbed fusaric acid, possibly gives rise to 3-*n*-butylpyridine, which is 100 times as toxic. Methylation—which, according to the variety of tomato, transforms 8–24% of the absorbed fusaric acid—has an opposite effect: a far-reaching detoxication of higher concentrations of fusaric acid. Thus, by the decarboxylation of a portion of the fusaric acid molecules, the tomato plant injures itself, and, by the methylation of another portion in other sites of the same cells, it protects itself from injury.

3) *Other conversion products of fusaric acid.*—In addition to the quaternary ammonium-base of fusaric acid amide, at least 2 neutral C^{14}-containing transformation or disintegration products of fusaric acid, whose identification is still open, can be detected in tomato cuttings 48 hours after the administration of the labeled fusaric acid (38).

Thus, quantitatively, the whole of the fusaric acid labeled in the C-atom of the carboxyl group can be followed up in tomato plants. In routine work, about 80% can be registered in the plant, but, in experiments designed for this purpose, as much as 90%

or more can be registered, about 10% passing into the air with the carbonic acid as the result of decarboxylation. For more exact identification of separate moieties—for example, the 3-*n*-methylpyridine—it would, however, be necessary to label the fusaric acid molecules at not only the C-atom of the carboxyl group but also at some other point.

THE INJURIOUS EFFECTS OF FUSARIC ACID ON THE WATER-PERMEABILITY OF PLANT PROTOPLASTS.—Two different processes are concerned in the intake of water by plant protoplasts: the familiar physical osmotic component, which obeys Fick's law of diffusion, and (only recently discovered) an active, non-osmotic component that is supported by the metabolism of the cells.

The rate of physical osmotic intake of water by the protoplasts depends, other things being equal, on the resistance to diffusion of their plasma membranes and their plasma, that is, on their "water permeability." Thus, in protoplasts whose water-permeability is impaired, the direction and amount of injury can be measured by the time they need (if they have previously been plasmolyzed) to revert to their original condition ("deplasmolyzation time").

The metabolically controlled, non-osmotic water intake is accomplished by the protoplasts, independently of the concentration gradient but at the expense of energy; hence, the protoplasts can maintain it only as long as the energy of respiration is available to them (5, 6, 25). Hence, *ceteris paribus*, impairment of cell respiration leads to a change in the rate of water intake.

This non-osmotic component of water intake receives little attention in phytopathological literature; thus, all toxigenic changes in the rate of water intake of protoplasts are alike attributed to corresponding changes in the water-penetrability of their plasma boundary layers and their plasma. In the present part, therefore, we discuss only the toxigenic changes in the osmotic water-permeability of protoplasts, and later—in relation to toxigenic impairments of the ferment systems of the host—we return to the possibility of a toxigenic effect on non-osmotic water intake.

Injurious effects of the metabolic products of wilt pathogens on the osmotic water-permeability of plant protoplasts were first studied by Thatcher (57). He put tomato cuttings in culture filtrates of *Fusarium lycopersici* and demonstrated that, when the first signs of wilt appeared, the water-permeability of the palisade cells in the leaves was about twice as high as in the corresponding palisade cells of healthy plants. Similarly, Gottlieb (23, 24) found that the fluid in the vascular bundles of tomato plants attacked by *Fusarium lycopersici* Sacc. increased the water-permeability of the medullary cells of tomato shoots almost threefold; the fluid must, therefore, contain substances that make the protoplasts more accessible to water. One of these toxic substances is fusaric acid, which we are interested in here.

The influence of increasing concentrations of fusaric acid on the water-permeability of plant protoplasts.—The injurious effect of fusaric acid on the water-permeability of plant protoplasts has been investigated in 2 model examples: in a green alga, *Spirogyra nitida* (Dillwijn) Link, and in the epidermal cells of the underside of the leaf of a flowering plant, *Rhoeo discolor* (L'Hér.). Hence, they serve as a substitute for tomato cells, which, on technical grounds, are ill-suited to deplasmolyzation experiments (19).

The measure of injury to the water-permeability of the protoplasts is the quotient of the deplasmolyzation times of the controls in tap water, divided by the deplasmolyzation times of the protoplasts injured by fusaric acid in given concentrations. Q-values over 1 thus mean a heightening of water-permeability, and Q-values under 1 mean a lowering. Where a "temporary" increase of the water-permeability of protoplasts is mentioned in this paper, "temporary" implies an increasing concentration of fusaric acid; the impairment of water-permeability produced by a specific concentration of fusaric acid is always irreversible. The full extent of irreversible impairment is already attained when the given fusaric acid solution has been acting for only 10 minutes (1).

1) *The threshold of impairment of water-permeability of protoplasts.*—Fusaric acid begins measurably to impair the water-permeability of protoplasts of *Rhoeo discolor* at a concentration of $10^{-9}M$ (Fig. 1A), of protoplasts of *Spirogyra nitida* at 10^{-8}, and the medullary cells of young tomato plants at 10^{-7}–$5.10^{-8}M$. The corresponding threshold values of dehydrofusaric acid for *Rhoeo* and *Spirogyra* are $10^{-8}M$ (1, 3, 19).

The fact that the susceptibility of tomato cells to fusaric acid is some 50 times less than that of *Rhoeo* may be determined not by the species but by the nature of the tissues tested—perhaps, for example, by a lower susceptibility of medullary cells than of epidermal cells. In the stems of angiosperms the permeability of the protoplasts diminishes from without: it is highest in the epidermal cells, considerably lower in the sub-epidermal cells, and lowest in the deeply lying parenchymatous layers and in the pith (30, 59).

Concentrations of fusaric acid that correspond to these threshold values of injury to the water-permeability of protoplasts are soon attained both *in vitro* and *in vivo*. A concentration of $10^{-9}M$, at which injury to the epidermal cells of *Rhoeo discolor* begins, corresponds to a fusaric acid content of the solution of 0.18 μg/l; a concentration of 10^{-7}–$5.10^{-8}M$, at which the medullary cells of young tomato plants begin to show injury, corresponds to a fusaric acid content of about 14 μg/l.

In culture filtrates of *Fusarium lycopersici* about 50 mg of pure, crystallizable fusaric acid per liter of nutrient solution can be detected even by the tenth day after inoculation of the flasks (54); this corresponds to a fusaric acid concentration of about

$3.10^{-4}M$. According to observations by Kern (35), the "*Ustilago-activity*" in culture filtrates of *Fusarium lycopersici*, which corresponds closely to the fusaric acid content of the solution, increases steadily during the first 2 weeks; the fusaric acid in the culture fluid therefore surpasses the threshold of injury to the water-permeability of *Rhoeo* and tomato cells, even on the first or second day.

This must also happen *in vivo*, inside the diseased host plants. Thus, in young cotton plants inoculated with *Fusarium vasinfectum* Atk., at least 17 mg of fusaric acid per kg of fresh weight can be detected 2 weeks after infection, that is, about 20 mg/l of tissue fluid. This is only a third less than *Fusarium lycopersici* forms in culture *in vitro* in the same period (19, 34). Hence, on the second day after infection, an average of at least 1–2 mg of fusaric acid per kg of tissue fluid is probably present in the cell sap of these plants. This average is about 100 times as great as the above-mentioned threshold of injury to medullary cells of tomato. The threshold of injury to the water-permeability of the host's protoplasts by fusaric acid is thus surmounted *in vivo* even at the inception of pathogenesis.

2) *Temporary increase in the water-permeability of protoplasts.*—When the threshold has been passed the water-permeability of the protoplasts is temporarily increased in all 3 test objects by the action of both fusaric acid and dehydrofusaric acid, and at a concentration of 10^{-6}–$10^{-7}M$ attains a peak somewhere about 1½–2 times the normal value (Fig. 1-AB). This initial increase in the water-permeability of the protoplasts takes place in the same way in all 3 test objects; correspondingly, these physiological injuries do not depend on a particular species.

If the increase of water-permeability in the protoplasts that Thatcher and Gottlieb established for tomato plants wilted by *Fusarium lycopersici* is due entirely to fusaric acid, the concentration of fusaric acid must have approximated 10^{-6}–$10^{-7}M$. With a further increase in the concentration of the test solution, the curve of injury changes its direction and returns to the original level (quotient 1.0) at a concentration of about 10^{-5}–$10^{-4}M$; here the protoplasts behave like the controls in tap water.

Thus, with increasing concentrations of fusaric acid, there is a threshold value where the fusaric acid does not yet impair the water-permeability of the protoplasts ($10^{-9}M$) and then a second range of concentrations (at 10^{-5} or 10^{-4}) where the water-permeability of protoplasts is no longer measurably impaired.

3) *The last phase of injury to the water-permeability of protoplasts.*—At still higher toxin concentrations, a new phase of injury to water-permeability begins (Fig. 1-AB); in contrast to the earlier concentration phase, the course of this second phase is determined by the species; that is, in *Spirogyra* it differs from that in the 2 examples from the realm of flowering plants.

This divergence is possibly connected with the fact that at least *Spirogyra* and *Rhoeo* (tomato tissues have not yet been tested in this respect) belong to different plasma types: *Spirogyra*, on account of its permeability to urea and methyl-urea, belongs to the amidophilic pore-type, whereas *Rhoeo* belongs to the amidophobic solution-type.

In *Spirogyra nitida* (Fig. 1B), fusaric acid concentrations above $10^{-5}M$ at first induce a transient tightening, or "waterproofing," of the protoplasts, and thereafter (above about $5.10^{-4}M$) a permanent increase in their water-permeability to about twice the normal value. The impairment of the water-permeability of *Spirogyra* protoplasts with increasing concentrations of fusaric acid goes through 3 phases; between 10^{-5} and $10^{-5}M$, an initial transient increase; between 10^{-5} and about 5.10^{-4}, a temporary "waterproofing"; and above $5.10^{-4}M$, a second definitive increase.

Rhoeo and tomato protoplasts (Fig. 1A) respond to higher fusaric acid concentrations in a manner exactly opposite to that of *Spirogyra* protoplasts: a marked decrease in water-permeability. With increasing fusaric acid concentrations the former pass through 2 contradictory phases of injury: first (between 10^{-9} and about $10^{-4}M$) a temporary increase, and thereafter a definitive tightening, or "waterproofing"; it looks as if the plasma membranes have been coagulated.

Dehydrofusaric acid in increasing concentrations injures the water-permeability of *Spirogyra* and *Rhoeo* protoplasts in the same way as fusaric acid, but in *Spirogyra* (Fig. 1B) the slight "waterproofing" in the region of $10^{-4}M$ is absent; further, in higher concentrations, dehydrofusaric acid is some 10 times as toxic to *Rhoeo* protoplasts as is fusaric acid (3).

Thus, it is characteristic of the action of increasing concentrations of fusaric acid or of dehydrofusaric acid on the water-permeability of plant protoplasts that the dosage-effect relationship changes its direction: lower concentrations increase the water-permeability of the protoplasts; at higher concentrations this increase diminishes and wholly disappears; at still higher concentrations there takes place, according to the subject concerned, either a fresh increase in water-permeability or a "waterproofing" of the protoplasts.

The constituents of the fusaric acid molecule active in the impairment of water-permeability.—In the alternating impairment of water-permeability by increasing concentrations of fusaric acid, 2 constituents of its molecule are concerned in different ways. The pyridine ring controls the increase of water-permeability at lower concentrations that occurs in the same way in all 3 test objects; and the *n*-butyl group in the β position is responsible for the species-specific impairment that begins at higher concentrations and in *Rhoeo* and tomato protoplasts leads to a definitive "waterproofing," but in *Spirogyra* protoplasts leads to a further increase in their water-permeability.

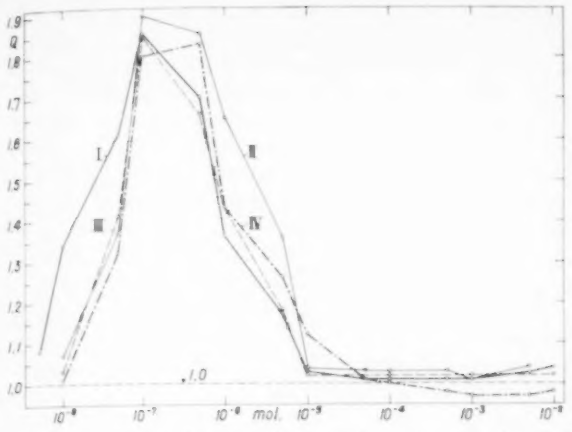


FIG. 2. The influence of increasing concentrations of pyridine (curve I), α-picolinic acid (curve II), 3-methylpyridine (β-picolin; curve III), and 5-methylpyridine-2-carbonic acid (curve IV) on the water-permeability of protoplasts of *Rhoeo discolor*. *Abscissae:* molar concentration of solutions. *Ordinates:* degree and kind of impairment of water-permeability; quotients above 1 indicate a heightening, quotients below 1 a lowering, of water-permeability. (After Bachmann (1)).

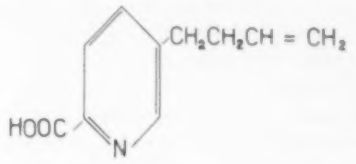
1) *The significance of the pyridine ring in the impairment of the water-permeability of protoplasts.*—The transient increase of water-permeability in the protoplasts of *Rhoeo*, tomato, and *Spirogyra*, in the range of concentration 10^{-8}–$10^{-5}M$, is induced not only by fusaric and dehydrofusaric acid (Fig. 1-AB) but also by pyridine alone (Fig. 2, curve I) and by some 20 different pyridine derivatives related to fusaric acid (e.g. Fig. 2, curves II-IV), whose sole constituent in common is the pyridine ring and which are differentiated from one another by their side-chains (1, 2).

2) *The significance of the side-chains in the impairment of the water-permeability of protoplasts.*—If the toxin concentration rises above the value of $10^{-5}M$, all the curves of impairment (in contrast to corresponding curves of fusaric and dehydrofusaric acid) remain in Fig. 2 at the level of the original base line. Thus, in higher concentrations, these substances no longer impair the water-permeability of protoplasts: the pyridine ring has exhausted its capacity, and the side-chains in question in Fig. 2—the carboxyl group in the α position (curve II), the methyl group in the β position (curve III), and their combination (curve IV)—are no longer toxic to the water-permeability of protoplasts.

The specific injury to the water-permeability of protoplasts by higher concentrations of fusaric and dehydrofusaric acid must, therefore, be determined by their *n*-butyl group in the β position. Indeed, the 3-*n*-butylpyridine, which differs from fusaric acid only in its lack of a carboxyl group, resembles fusaric acid in possessing at higher concentrations a "waterproofing" effect on *Rhoeo* protoplasts and a capacity to increase the water-permeability of *Spirogyra* protoplasts (Fig. 1C). The same effects occur with higher concentrations of all the pyridine derivatives possessing a similar side-chain that have been tested, whereas they are absent in the homologous compounds lacking this side-chain. For example, if the butyl group of fusaric acid (5-*n*-butylpyridine-2-carbonic acid) is replaced by a methyl group (5-methylpyridine-2-carbonic acid) the acid, at higher concentrations, loses its specific effect on the water-permeability of protoplasts (Fig. 2, curve IV).

The pathogenic action of the aliphatic side-chain increases at first with the length of the chain (Fig. 3); the peak of toxicity is more or less attained with 3-*n*-butylpyridine: 3-*n*-amylpyridine and 3-*n*-hexylpyridine produce no appreciably heavier "waterproofing" of the protoplasts (1). This increase of toxicity with growing length of the aliphatic side-chain is possibly connected with an increase of lipoid solubility, and parallel to this with an increase in capacity to penetrate into the protoplasts.

If, by the transformation of fusaric acid into dehydrofusaric acid, the 3-*n*-butyl group, other things being equal, remains unsaturated—



Dehydrofusaric acid

—its pathogenic effect on the protoplasts of *Spirogyra* and *Rhoeo* is altered qualitatively or quantitatively, as mentioned before.

The significance of the other side-chains to the impairment of the water-permeability is still obscure. Thus, the carboxyl group in the α position modifies the toxicity of the pyridine ring as well as that of

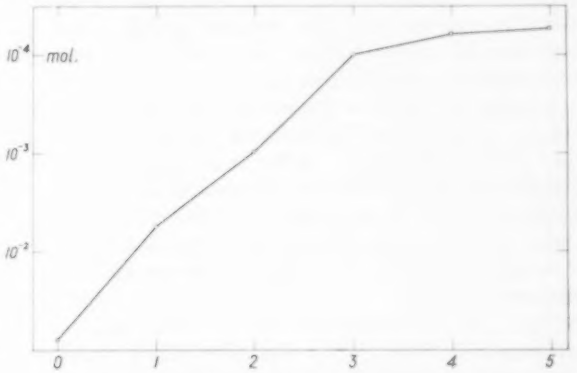


FIG. 3. The dependence of the "waterproofing" action of different pyridine derivatives on the length of their aliphatic side-chain in the β position. *Abscissae:* number of CH_2 groups in the side-chain. 0 = 3-methylpyridine. 1 = 3-ethylpyridine. 2 = 3-*n*-propylpyridine. 3 = 3-*n*-butylpyridine (see Fig. 1C). 4 = 3-*n*-amylpyridine. 5 = 3-*n*-hexylpyridine. *Ordinates:* concentration necessary to reduce the water-permeability of *Rhoeo* protoplasts to about 60%. (After Bachmann (1)).

the aliphatic side-chain; for example, 3-*n*-butylpyridine, which differs from fusaric acid only by its lack of a carboxyl group in the α position, is about 100 times as toxic to the protoplasts of *Spirogyra* and *Rhoeo* (Fig 1C) as is fusaric acid at both lower and higher concentrations (Fig. 1-AB).

The influence of nutrition on the impairment of water-permeability by fusaric acid.—Impairment of the water-permeability of *Rhoeo* protoplasts by increasing concentrations of fusaric acid, described in the preceding sections, holds good only for normally nourished plants; overfeeding or underfeeding with nitrogen, phosphoric acid, or potassium can greatly alter the type of reaction to the 2 active groups of the fusaric acid molecule (13).

An eightfold overdose of any of the 3 nutritive substances will heighten some 10–100 times the capacity of *Rhoeo* protoplasts to resist the *pyridine ring* of fusaric acid. In order to impair the water-permeability of their protoplasts to the degree shown by normally fed specimens, plants given 8 times the normal amount of nitrogen or potassium require 100 times as much fusaric acid, and plants given 8 times the normal amount of phosphoric acid need 10 times as much fusaric acid (Fig. 4; left portions of curves 8N and 8P, respectively).

Underfeeding with one of these 3 nutritive substances produces divergent results. The sensitivity of *Rhoeo* protoplasts to the pyridine ring of fusaric acid is not influenced by underfeeding with potassium (even in amounts less than $\frac{1}{64}$ of the normal ration), but only by a deficiency of nitrogen or phosphoric acid; both completely extinguish the sensitivity of the *Rhoeo* protoplasts to the pyridine ring of fusaric acid—nitrogen deficiency at $\frac{1}{8}$ and phosphoric acid deficiency at $\frac{1}{64}$ of the normal ration (Fig. 4; left portions of curves $\frac{1}{8}$N and $\frac{1}{64}$P, respectively). It is as if these nitrogen-deficient or phosphoric-acid-deficient *Rhoeo* protoplasts are no longer able to synthesize the specific receptors for the pyridine ring of fusaric acid, although their water-permeability seems, to our eyes, to remain normal.

Because of this exclusion of the pyridine ring, fusaric acid in lower concentrations has no effect in the sector "impairment of the water-permeability of protoplasts" in plants underfed with nitrogen or phosphoric acid; in these plants, toxic effects show themselves only if the concentration threshold for the injurious action of the aliphatic side-chain is overstepped.

The type of reaction of *Rhoeo* protoplasts to the aliphatic side-chain of fusaric acid, which in higher concentrations controls the impairment of water-permeability, is influenced slightly or not at all by over- or underfeeding with nitrogen or potassium, and by underfeeding with phosphoric acid (Fig. 4; right portions of the curves $\frac{1}{8}$N, 8N, and $\frac{1}{64}$P). The reaction type is affected only by eightfold overfeeding with phosphoric acid, which reverses the direction of action of the aliphatic side-chain: the aliphatic side-chain of fusaric acid no longer diminishes the water-permeability of *Rhoeo* protoplasts, but increases it (Fig. 4; right portion of curve 8P).

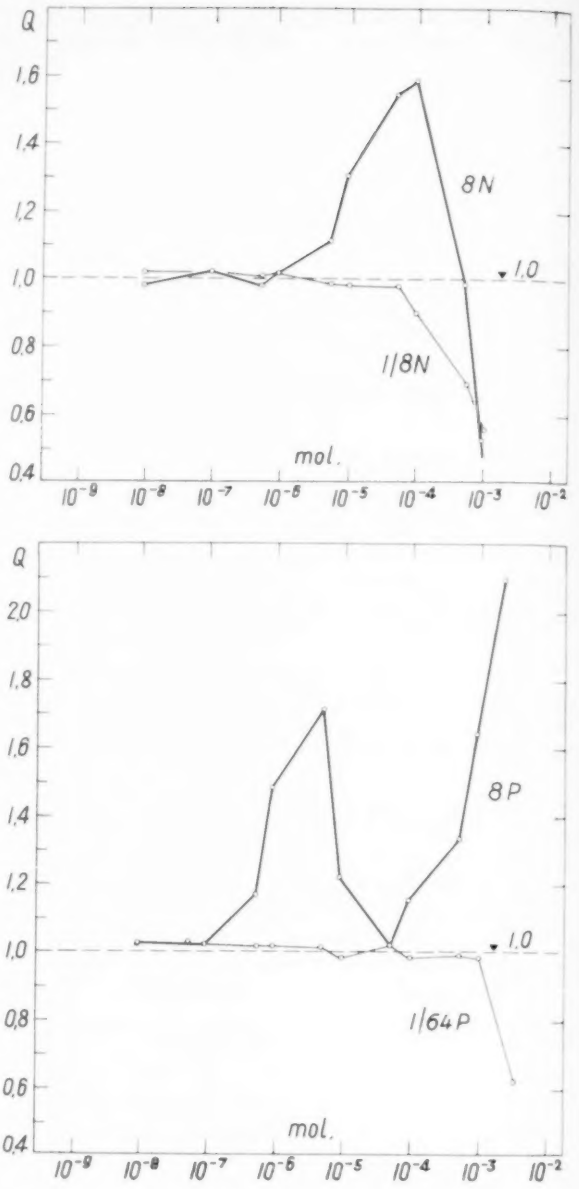


FIG. 4. The influence of varying nitrogen or phosphoric acid nutrition on impairment of the water-permeability of protoplasts of *Rhoeo discolor* by increasing concentrations of fusaric acid. Curves 8N and 8P show eightfold overfeeding with nitrogen and phosphoric acid, respectively. Curves 1N and 1P (normal nutrition) are given in Fig. 1A, curve *Rhoeo*. Curves $\frac{1}{8}$N and $\frac{1}{64}$P represent nitrogen and phosphoric acid rations reduced, respectively, to $\frac{1}{8}$ and $\frac{1}{64}$ of the normal ration. *Abscissae:* molar concentration of fusaric acid solutions. *Ordinates:* amount and kind of injury to water-permeability; quotients above 1 indicate a heightening, quotients below 1 a lowering, of water-permeability. (After Gäumann and Bachmann (13)).

The water-permeability of *Rhoeo* protoplasts over-

fed with 8 times the normal amount of phosphoric acid is therefore doubly impaired in the same way by increasing concentrations of fusaric acid: at lower concentrations it is increased by the pyridine ring, and at higher concentrations by the aliphatic side-chain. A curve of similar shape has been observed for *Rhoeo* protoplasts only in reaction to increasing concentrations of coramine, the diethyl-amine of nicotinic acid (2); on the other hand it is of frequent occurrence in *Spirogyra* (e.g. Fig. 1B).

How combinations of such anomalies—for example, nitrogen excess with phosphoric acid deficiency—work is in this respect still unknown. In natural disease, the concentration of fusaric acid is almost always higher in the neighborhood of the focus than in the distal parts of the host; hence, one and the same nutritional anomaly may act differentially on the toxin sensitivity of a host's protoplasts in the neighborhood of the focus and in distal tissues. Because the nutritional state of the host alters its sensitivity to other fields of action of fusaric acid in a different way, the influence of nutrition on the *visual symptoms* of disease obeys its own laws (63).

THE IMPAIRMENT OF THE SEMI-PERMEABILITY OF THE PLASMA MEMBRANES BY FUSARIC ACID.—As described earlier, fusaric acid travels quickly up tomato cuttings so that nearly one third of the dose administered can be detected in the leaves after 2 hours. At this time, impairment of the semi-permeability of the plasma membranes in the leaves becomes apparent: their cuticular recrement, measured by the electrical conductivity of the transpiration residues, now begins to increase, sharply during the first 5 hours and more slowly thereafter (41).

The first macroscopically recognizable wilt symptoms, under the conditions of the experiment, appear only 10–12 hours after application of the toxin: at earliest, 8 hours after the beginning of the pathological increase of the cuticular recrement. Thus, the obvious impairment of the semi-permeability of the plasma membranes anticipates the appearance of visual symptoms of the disease by at least 8 hours.

As shown in Table 1, the potassium ions are chiefly concerned in this pathological recrement, which may increase to over 300 times the amount of the value in the controls; the sodium and calcium ions are involved to a lesser extent. Fusaric acid thus injures not only the general salt-permeability of the plasma membranes, but also their selective permeability for specific ions.

In addition to these ions, amino acids (but no measurable quantities of sugars) pass out from within the cells into the transpiration stream of the cell walls and are carried by this to the surface of the leaf. Since free amino acids are present in the plasma in only small amounts, the amino acids exuded (see Table 1) must derive from a partial lysis of plasma proteins; larger fragments (peptides) sometimes become apparent in the excretions. This recrement of amino acids, etc., has therefore the character

TABLE 1.—*Composition of the pathological recrement on the upper surface of tomato leaves as a reaction to fusaric acid and lycomarasmin, respectively—in mg/100 cm² leaf area for cations and in μg/100 cm² leaf area for amino acids (After Linskens (41))*

Substance	Fusaric acid	Lycomarasmin	Controls (healthy plants)
Na	0.81	0.10	0.02
K	3.45	0.22	0.01
Ca	0.38	0.47	0.05
Glycine	69.0	88.0	29.0
Alanine	29.8	31.8	15.4
Serine	46.2	34.8	11.8
Glutamic acid	18.4	11.0	6.7
Proline	32.0	40.7	7.4
Thyrosine	21.2	23.6	9.2
Leucine	45.5	52.8	11.2

of a partial elimination of necrogenous decomposition products from the injured host cells (41).

With this impairment of the semi-permeability of the outer plasma membrane (the plasmalemma) and of the tonoplast, the physical conditions of osmotic pressure—and therewith of turgor—are destroyed. Turgescence in plant tissues depends on the separation, by a semi-permeable membrane, of a transpiration stream low in salt content that circulates in the cell walls, from an osmotically efficient cell content. If this semi-permeability is impaired to such a degree that metallic ions, amino acids, and even peptides escape into the transpiration stream circulating in the cell walls, and if, in consequence, osmotically active substances are present on both the inner and outer sides of the outer plasma membrane, then turgor is no longer possible. The affected tissues wilt because their protoplasts are "turgor-sick"; and, as we shall see later, they wilt even if they have ample water at their disposal.

Impairment of the polar permeability, of the outer plasma membrane especially, does not merely open a way for the large molecules of the host from the inside of the cells into the transpiration stream in the cell walls. It also opens a way from the cell walls inward (to the inside of the host's protoplasts) for the large molecular metabolic products of the pathogen that are brought to the cells in the transpiration stream: its toxins, enzymes, and accompanying substances. It is scarcely to be expected that resistance to diffusion in only one direction could still exist in protoplasts whose water-permeability and polar permeability have been wholly destroyed by toxin.

An impairment of the polar permeability of tomato protoplasts, similar to that due to fusaric acid, is also produced by lycomarasmin (Table 1, column 3). Inside the host these 2 toxins reinforce each other's injurious action on the polar permeability of the protoplasts, just as, under certain conditions, they reinforce each other's impairment of water-permeability (14).

Thus, 2 chemically different toxins, a pyridine-carbonic acid and a dipeptide, by different mechanisms lead to the same injury: an abolition of the

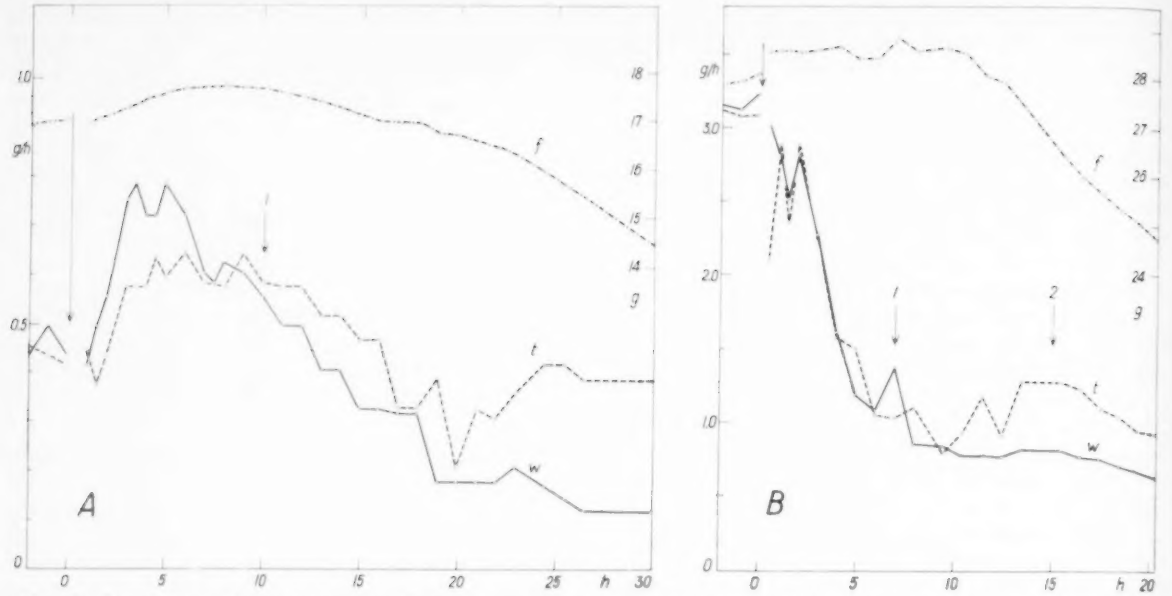


FIG. 5. Impairment of the water economy of a cut tomato shoot by fusaric acid: A) slow poisoning (about 20 mg of toxin per kg fresh weight taken up during the first hour of experiment); B) gross poisoning (about 200 mg of toxin per kg fresh weight taken up during the first hour). *Abscissae:* hours from start of experiment (indicated by arrow). *Ordinates: w*, fluid intake; *t*, cuticular and stomatal transpiration of the whole shoot, both in g/hour. *f*, fresh weight of shoot in g. *1*, grooves in stem; *2*, collapse of plant. (After Gäumann, Kern, Schüepp, and Obrist (18)).

polar permeability of the protoplasts. This convergence of effects makes it possible to understand why different types of wilt pathogen can, with different toxins, produce similar disease pictures and similar courses of disease. In our first paper we discussed an example of this in tomato wilt (12, p. 350).

IMPAIRMENT OF THE WATER ECONOMY IN TOMATO SHOOTS BY FUSARIC ACID.—In the experiments to be described here, the water economy of cut tomato shoots was examined by automatic recording of their water intake and of their cuticular and stomatal transpiration, both under constant external conditions and under the influence of a given toxin concentration. Since it is difficult to alter the water economy of plants by toxigenic factors, this method is some 5000 times less sensitive than the toxic impairment, described earlier, of the water-permeability of single tomato protoplasts.

If the dose of fusaric acid is strong enough to induce visible symptoms, particularly small necroses, the appearance of the first manifest symptoms will indicate a turning point in impairment of the water economy.

In the pre-manifest, symptomless phase—during the "incubation period" of the toxin, as it were—the shape of the curve will largely be determined by the degree of poisoning. With a slow poisoning of tomato shoots, such as always occurs in natural disease (as with the intake of about 20 mg of fusaric acid per kg of fresh weight, during the first hour of experiment, Fig. 5A), the water exchange in the shoots is at first greatly increased. On the other hand, in gross poisoning of the shoots, as with the supply of about 200 mg of fusaric acid per kg of fresh weight during the first hour of the experiment (Fig. 5B), the entire water exchange is immediately crippled and, within 3–5 hours, is sharply reduced, to less than one half. However, in both degrees of poisoning, the curves of water intake and water output lie close together; hence, during the pre-manifest phase of injury, the experimental plants suffer no loss of weight.

The decisive change in their water economy coincides with the appearance of the first necroses, etc. The water exchange in both degrees of poisoning is now further diminished (although, in general, more slowly than before), but the curves of water intake and water output diverge from one another so that transpiration becomes greater than fluid intake; hence, the experimental shoots lose moisture in increasing amount, and, later, turgor also. Thus, in fusaric acid poisoning, wilting as a consequence of water loss sets in only after the appearance of visible injuries (18).

The mechanism is not yet known by which fusaric acid gives rise to these successive impairments of the water economy in tomato shoots. One area of its field of activity—the lowering of water exchange—may perhaps be related to its capacity, in similar concentration, to reduce the water-permeability of protoplasts. Some pyridine derivatives, which in the tested concentrations "waterproof" the protoplasts of *Rhoeo discolor*, also lower the water exchange in tomato shoots. Conversely, other pyridine derivatives, which in the given concentrations have no effect on the water permeability of *Rhoeo* protoplasts, also do not impair the water economy of tomato shoots.

But there are exceptions: α-picolinic acid, in certain concentrations, has no action on the water-perme-

ability of *Rhoeo* protoplasts (Fig. 2, curve II), but nevertheless impairs the water economy of tomato shoots in a characteristic way; hence, the fact that it lowers the water exchange must depend on mechanisms of impairment other than those that induce "waterproofing" of protoplasts. Further, the temporary increase of water exchange in the slow poisoning of tomato shoots (Fig. 5A) cannot be explained by any toxic impairments of the water-permeability of protoplasts that are known thus far.

INJURY TO THE FERMENT SYSTEMS OF THE HOST BY FUSARIC ACID.—Our concepts about disturbance of the enzyme systems of the host are based, for the most part, on hypothetical grounds; hence, the relevant problems might be described more adequately by a biochemist interested in plant pathology than by a botanist.

The withdrawal of heavy-metal ions by chelation.—Because of its chemical configuration, fusaric acid possesses the capacity to release, in the host cells, heavy-metal ions from their natural combinations and to form with them (about 1 heavy-metal ion to 1 molecule of fusaric acid) complexes having a cyclic structure—chelates (10).

By this withdrawal of heavy-metal ions, fusaric acid interferes with many of the components and vital processes within the cell; thus it impairs the structure of the protein framework of the marginal layers of the plasma—and the plasma itself—thereby altering their permeability. Possibly it is also able to withdraw the calcium ions from the calcium pectate of the middle lamellae—as Bennet-Clark (4, p. 290) surmised for heteroauxin—and thus to transform the water-insoluble calcium pectate into water-soluble pectin compounds, readily accessible to the pectolytic enzymes of the pathogen.

Further, by the chelation of heavy-metal ions, the fusaric acid molecules interfere with many ferment systems of the host; the effects differ according to the site at which chelation takes place.

If it occurs, for example, in the stem and petioles of the host, and if only small amounts of toxin are involved, then, as a rule, visible injuries do not appear. The fusaric acid molecules, however, forward the heavy-metal ions into the leaf tissues—and there lose them again, possibly because of a change of pH or because of photochemical processes. According to the state of equilibrium in which the different enzyme systems of the host find themselves in relation to the ions in question, the results of this "vehicle-effect" are contrasting. With a sub-optimal content of the given metals, the additional ions brought into the leaf tissues promote the activity of the constructive or destructive enzymes. Where the heavy-metal content is high, the introduction of additional ions into the leaves provokes injuries, due to excess, as was demonstrated experimentally in the case of iron ions and lycomarasmin, which behaves similarly in this respect (15).

On the other hand, if the chelation of the heavy-metal ions occurs at the site of the specific activity of fusaric acid—that is, in the protoplasts of the leaf cells—it has an opposite effect: it immediately causes local deficiencies. The withdrawal of magnesium impairs both photosynthesis and, among other effects, the activity of pyruvic decarboxylase, whereas the withdrawal of manganese impairs the action of certain peptidases. Further, the activities of cytochrome oxidases and catalase are reduced by the withdrawal of iron, that of dipeptidase by the withdrawal of cobalt, and that of phenol oxidase and ascorbic oxidases by the withdrawal of copper, etc. Corresponding disturbances of enzyme activities by fusaric acid have been observed in rice plants by Tamari and Kaji (55, 56).

By the withdrawal of certain heavy-metal ions on one hand, and, on the other hand, by a local increase of other metal ions as a result of the "vehicle-effect," the ion balance in the different zones of activity of the host cells can be disturbed so that ion antagonism results (e.g. Mn:Fe, Cu:Mn).

All these effects of fusaric acid occur in the different tissues of the one host and in the different zones of activity of one and the same cell, side by side at the same time, and interact in many different ways. Some of them are of little vital importance, but others are decisive. From this obscure interplay of forces we shall select only 2 functional disturbances: impairment of the respiration of the protoplasts by fusaric acid, and, consequent upon this, impairment of their non-osmotic water intake.

The impairment of cellular respiration by fusaric acid.—If tomato plants are inoculated with *Fusarium lycopersici*, their foliar respiration shows an increase by the second day (8, 44). On the fourth day after infection it reaches a maximum at 126% of the normal value, and then declines steadily during the incubation phase (the first symptoms appear only on the eighth day after infection) and falls to 65% by the seventeenth day (44).

The respiratory increase at the start of pathogenesis is probably induced by lycomarasmin in cooperation with chelated iron encountered on its route; the lycomarasmin-iron complex causes a similar respiratory increase in cut tomato leaves during the first 2–6 hours of its activity, and later an inhibition (44). On the other hand, if isolated tomato mitochondria are subjected to increasing concentrations of lycomarasmin (Fig. 6), their succinic oxidase activity is at first greatly stimulated, and then inhibited (47, 48).

Fusaric acid, however, inhibits respiration from the beginning in tomato plants (44, 45, 63), rice plants (55, 56), and in *Saccharomyces cerevisiae* Hans. (51). In the last the inhibition is reversible during the first 2–3 hours; thus, it takes at least that time for decisive impairment of the mechanisms or their substrate to set in. Thereafter, the reversible moiety of the respiratory inhibition steadily diminishes, and after 45 hours it amounts to only 18%

(51). If isolated tomato mitochondria are subjected to rising concentrations of fusaric acid, their succinic oxidase activity is also inhibited from the beginning (Fig. 6); Fig. 1 and 2 suggest that the *n*-butyl group in the β position may play a rôle here.

The addition of small amounts of cytochrome C to the reaction mixture destroys the inhibitory effect of both toxins. Consequently, both lycomarasmin and fusaric acid interfere, in the range of the cytochrome C, with the oxidative decomposition of succinic acid —either directly, by arresting the cytochrome oxidase systems; or indirectly, by impairing the semipermeability of the mitochondrial membrane so that cytochrome C escapes and is lost to the mitochondria (47, 48).

In the course of natural disease, still other metabolic products of the pathogen probably interact on respiration; thus, in addition to fusaric acid, *Fusarium lycopersici* forms succinic acid (20, 21), and *Fusarium vasinfectum* forms ascorbic acid (31, 32, 33), both being substances that are probably involved in a cycle of respiration.

Impairment of the non-osmotic water intake of protoplasts by fusaric acid.—The impairments of respiration by fusaric acid just discussed affect the water intake of protoplasts both in its physical-osmotic and in its metabolically determined non-osmotic components—the former indirectly, and the latter directly.

The physically determined osmotic water intake is a matter of diffusion; but its prerequisite conditions depend on the vital activity of the cells. Impairment of the enzyme systems of the host cells by fusaric acid makes it more difficult for them to regulate the protein framework of their plasma membranes and their plasma, which has been thrown into disorder by the withdrawal of heavy-metal ions.

In contrast, the metabolically determined non-osmotic water intake of the protoplasts is directly affected by the toxigenic impairment of respiration, because it is just this respiration that supplies the energy for this form of water intake. The 2 active components of the fusaric acid molecule—the pyridine ring and the *n*-butyl group in the β position—impair the non-osmotic water intake in different ways, from which it may be concluded that they intervene at different points in the respiration cycles.

If one arrests the cytochrome oxidase in *Rhoeo* protoplasts by means of sodium azide and then subjects them to increasing concentrations of fusaric acid, the water intake at lower toxin concentrations (Fig. 6A) shows the marked rise characteristic of the pyridine ring. On the other hand, at concentrations above $10^{-5}M$ the lessening of water intake determined by the *n*-butyl group is mostly absent. The *n*-butyl group in the β position has, therefore, been rendered inactive by the previous blocking of the cytochrome oxidase; hence, in some way it impairs, as in isolated tomato mitochondria (Fig. 6, curve *Fs*), this enzyme in the *Rhoeo* protoplasts.

On the other hand, if one arrests oxidative phosphorylation in the *Rhoeo* protoplasts by 2,4-dinitrophenol, and again subjects them to increasing concentrations of fusaric acid, the increase of water intake conditioned by the pyridine ring is absent at lower concentrations (Fig. 6B). Only at concentrations above $10^{-4}M$ does inhibition of water intake, due to the *n*-butyl group, set in. Thus, the pyridine ring of fusaric acid has been inactivated by the previous blocking of the oxidative phosphorylation; hence, one may surmise that this ring in some way disturbs the oxidative phosphorylation of the *Rhoeo* protoplasts (1).

Thus, fusaric acid impairs the energy metabolism and, in consequence, the non-osmotic water intake of *Rhoeo* protoplasts by different mechanisms in lower and higher concentrations. Comparable differences in "choice" of point of impact—in this case determined by the host—were discussed in connection with the influence of nutrition on the water-permeability of protoplasts.

General considerations.—In the course of natural disease the mechanisms discussed in this paper undergo still further complications.

The cooperation of fusaric acid with other toxins of the pathogen.—All the plant-pathogenic fungi so far examined that form fusaric acid produce—besides

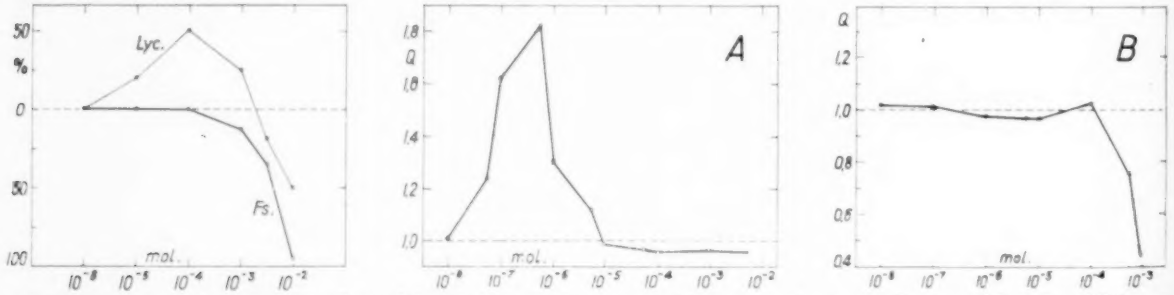


Fig. 6. Left graph shows the effect of increasing concentrations of lycomarasmin (curve *Lyc.*) and fusaric acid (curve *Fs.*), respectively, on the succinic oxidase activity of isolated mitochondria of tomato. *Abscissae:* toxin concentration. Positive *ordinates* indicate a stimulation, negative *Ordinates* an inhibition, of enzyme activity. The center and right graphs show the impairment of the non-osmotic water intake of the protoplasts of *Rhoeo discolor* by increasing concentrations of fusaric acid if, before treatment with fusaric acid, A) the cytochrome oxidase or B) oxidative phosphorylation in the *Rhoeo* protoplasts has been arrested. *Abscissae:* molar concentration of fusaric acid. *Ordinates:* degree and type of injury to the water-permeability. Quotients over 1 indicate a heightening, quotients below 1 a lowering, of water-permeability. (After Paquin and Waygood (48), slightly schematized, and after Bachmann (1)).

this and the usual non-specific enzymes, etc.—a number of other more or less specific toxins and associated substances; *Fusarium lycopersici*, for example, produces at least 6 (12, p. 350).

Fusaric acid, therefore, develops its pathogenic efficiency under different conditions—according to the order in which these substances are formed by the given pathogen, their threshold values within the various tissues and organs of the host, and the synergisms and antagonisms that occur there between them. For example, both fusaric acid and lycomarasmin are able to chelate heavy-metal ions (10); thus, in this respect, they compete with one another in the host tissues. Since *Fusarium lycopersici*, at the start of its growth, forms more fusaric acid than it does lycomarasmin, fusaric acid presumably deprives the lagging lycomarasmin of certain heavy-metal ions in the early stages of incubation (provided the chelating power of the 2 substances is equal under the conditions inside the cell). However, a few days later, because of the lower stability of the fusaric acid molecule, the situation may perhaps be reversed.

The pathogenic activity of fusaric acid is, therefore, far more complicated than is, for example, that of the toxin of *Pseudomonas tabaci* (Wolf & Foster) Stev., the pathogen of tobacco wildfire. This toxin, a structural analog of methionine (7, 62), is the only decisive toxin formed by this pathogen, and it therefore acts by itself within the host.

The range of associated toxins, etc., within which fusaric acid produces its results in natural infections is wide and of labile equilibrium, and further, fusaric acid itself has multiple possibilities for the working out of its own pathogenic effects. Here we shall discuss 2 aspects: the number of "targets" that can be hit by the fusaric acid molecules inside the cell and the orientation of the injury caused by increasing concentrations of fusaric acid.

The pleiotropic (manifold) injurious activities of fusaric acid.—The toxin of *Pseudomonas tabaci* interferes solely with the formation of certain amino acids, and, in particular as an antimetabolite, it inhibits the metabolism of methionine in the host: its entire lethal action rests on this one circumscribed effect. Again, diphtheria toxin (also probably through metabolic antagonism) merely arrests the synthesis of one or more components of the cytochrome system (46). Because of the extremely specialized functioning of these 2 toxins, their sites of activity within the host cells are narrowly confined to definite parts—the former to that structural moiety concerned with the synthesis of particular amino acids, and the latter to that which directly or indirectly governs cellular respiration.

With fusaric acid the situation is different. Chemically, its molecules are little differentiated and can, therefore, be introduced into the most varied functional and structural components of the host. Not only the molecule as a whole takes part in the injurious action but, in addition, some of its chemical constituents intervene on their own and independently of one another in the vital activities of the host, injuring, for example, different enzyme systems. Thus, in the same *Rhoeo* protoplasts, the pyridine ring of the fusaric acid impairs oxidative phosphorylation, and the aliphatic side-chain in the β position blocks the cytochrome oxidase.

Each of these injuries is specialized, as in the case of the above-mentioned toxins, to particular mechanisms of the host cells; but the number of possibilities of attack is greater with fusaric acid than with the above-mentioned toxins: fusaric acid can attack in many and divers ways, "veering in many directions." Its field of action is *pleiotropic*. Thus, like diphtheria toxin A, it impairs the cytochrome system of the host cells but also (for example, by the chelation of their heavy-metal ions) many other ferment systems of the host. In addition it impairs the water-permeability of the protoplasts, the semi-permeability of the plasma membranes, etc.

The sites of action within the host cells are as numerous as the objectives; thus, the receptors for impairment of semi-permeability are chiefly in the boundary layers of the plasma, whereas those for the impairment of respiration are in the mitochondria, etc.

Associated with this wide action spectrum of fusaric acid is its low stability. How far does it operate within the host cells in its intact state, and how far through its transformation and decomposition products? In certain zones of the cell it is methylated, and thereby detoxicated in higher concentrations, for example, in relation to the impairment of the water-permeability of protoplasts. In other zones of activity of the same cell it is decarboxylated at the same time, and its toxicity in the same sphere of action is thereby increased about 100 times. Other transformations and decompositions perhaps further widen its pleiotropic field of action and open possibilities of inflicting injury that are inaccessible to the intact fusaric acid molecule.

In the diverse organisms of the wide action spectrum of fusaric acid—which ranges from the bacteria, algae, and fungi to the flowering plants—the derangements to which it can give rise probably differ in importance. One can imagine that certain injuries of more fundamental type—for example, inhibition of the respiratory systems of the cells—possess a wide action spectrum and affect more or less all the organisms and organs susceptible to fusaric acid, and that such generalized injuries will be overlain by additional host-specific or organ-specific disturbances. This host- or organ-specificity of the secondary effects is confirmed by the fact that high concentrations of fusaric acid alter the water-permeability of *Rhoeo* and tomato protoplasts in opposite directions, as they do in *Spirogyra* cells (Fig. 1-AB). Moreover, fusaric acid provokes diverse visible symptoms of injury in the different experimental plants, and, at times, in different tissues of a single plant (12, p. 351).

Orientation of the harmful effects from increasing concentrations of fusaric acid.—The effectiveness of certain biologically active substances increases in proportion to their rising concentration (that is, in a straight line or logarithmically); this form of behavior may be termed the *isodromic (continuous)* type.

With other substances the direction of injury is reversed when a given threshold of concentration is surmounted (for example, stimulation-paralysis). This form of behavior may be termed the *enantiodromic (anatropous)* type.

An *isodromic increase of activity* with rising concentrations of fusaric acid may, for instance, be conjectured where fusaric acid removes the heavy-metal ions from an enzyme system and thereby deactivates it: the more the molecules in this enzyme lose their heavy-metal ion, the greater will be the paralysis of the whole system.

An *enantiodromic course of injurious activity* with rising concentrations of fusaric acid is discussed above, e.g. in relation to the impairment of the water-permeability of *Rhoeo* and tomato protoplasts: low concentrations increase their water-permeability; higher concentrations reduce it.

These complicated dosage-effect relationships give rise to an obscure situation in the diseased plant. The level of fusaric acid generally diminishes from the focus to the distal parts, and from the "privileged" to the "non-privileged" tissues. Moreover, it permanently changes during the course of the disease. For this reason, fusaric acid produces isodromic and enantiodromic (stimulating and paralyzing) effects at the same time in the different tissues of a shoot and in the different zones of activity, side by side in one and the same cell. The example of the impairment of the water-permeability of protoplasts may illustrate the consequences.

At the onset of pathogenesis the fusaric acid concentration in the focal region is low, perhaps 10^{-6}–$10^{-5}M$; consequently, the pyridine ring of the fusaric acid produces there an increase in the water-permeability of the protoplasts. But, within a few days, the fusaric acid concentration at the focus may rise to about $10^{-3}M$, so that the water-permeability of the host protoplasts is now reduced to about half its normal level by the aliphatic side chain.

In distal parts of the host the conditions for fusaric acid concentration develop in a different way. The "privileged" leaf tissues specifically concentrate the fusaric acid (37), so that the aliphatic side chain reduces the water-permeability of their protoplasts. In the "non-privileged" host tissues the fusaric acid may be present in considerable dilution, perhaps only in a strength of 10^{-6}–$10^{-5}M$, so that its pyridine ring may possibly double the water-permeability of these protoplasts.

Thus, even within a single sphere of activity, the summation of the injurious effects of fusaric acid within a diseased plant is highly complex.

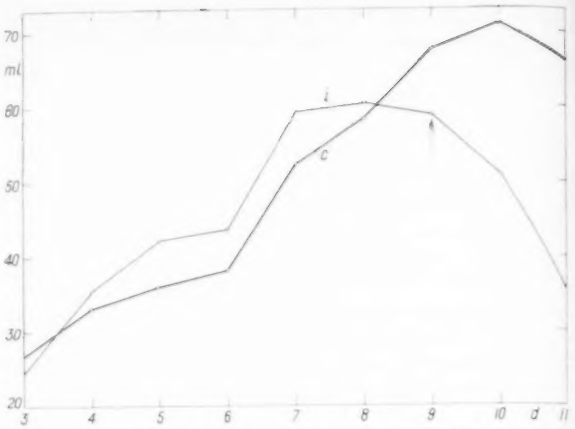


FIG. 7. The curve of transpiration in rooted (growing) tomato plants at the beginning of disease due to *Fusarium lycopersici*. *Abscissae:* days after infection. *Ordinates:* water output in ml per 1000 cm^2 of leaf surface. *i*, infected plants; *c*, healthy control plants. *Arrow:* appearance of first symptom of disease (a foliage leaf starts to wilt). (After Scheffer and Walker (53)).

THE COOPERATION OF WILT-INDUCING FACTORS IN NATURAL DISEASE.—The syndrome of pathological wilting is, in our opinion, induced by a combination of direct and indirect injuries. In direct injuries the protoplasts themselves become diseased, thereby initiating a wilting in that part of the shoot; indirect injuries arise mainly from irregularities of water distribution: the protoplasts are healthy in themselves but lose turgor because their water supply is insufficient.

Loss of turgor following toxigenic injury to the protoplasts in wilting tissues.—The toxins formed by the pathogen (fusaric acid and lycomarasmin, for example, in tomato wilt) are carried by the transpiration stream into the leaves, where they give rise to toxigenic injury of the protoplasts; this at first leads to an increase in transpiration, as observed in tomato wilt by Ludwig (42) and Scheffer and Walker (53; our Fig. 7).

In the case of fusaric acid this increase in transpiration is probably due to a general raising of the water exchange (Fig. 5A). In the case of lycomarasmin it is perhaps based on impairment of the water-holding capacity of the protoplasts: lycomarasmin, by its chelating ability, breaks the cation links between the protein chains, thereby increasing the solubility of the plasma, and correspondingly lessening its water-holding capacity. A model example is that of Harkness and Wassermann (26), who subjected actomyosin to ion exchanges and showed that their water solubility increased with reduction of their ash content. Because of this impairment of the water-holding capacity of the protoplasts, lycomarasmin, in a given concentration range, is able to induce toxigenic water loss in tomato shoots without toxigenic wilting (Fig. 8A).

This initial heightening of transpiration in the diseased plant is probably without significance as

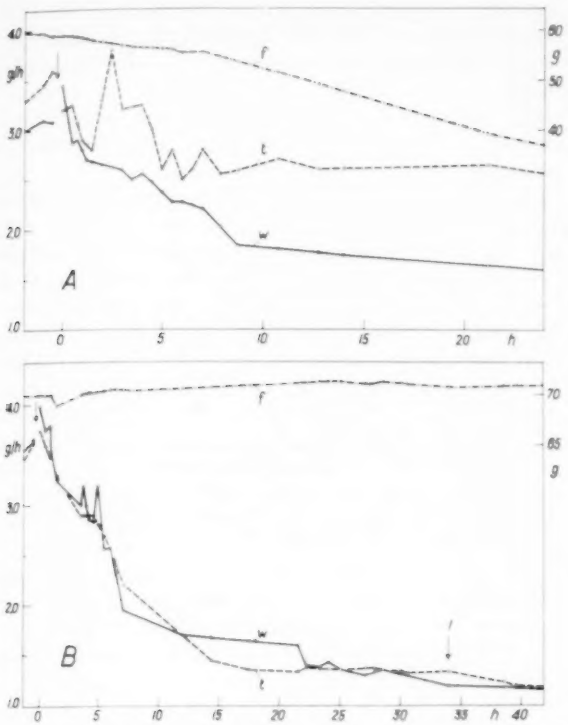


FIG. 8. A) Toxigenic water loss without pathological wilting; this occurs in tomato shoots when they are given lycomarasmin in high dilution ($10^{-5}M$). B) Toxigenic wilting without water loss; this occurs in tomato shoots if the wilt experiment is carried out in an atmospheric humidity approaching saturation (relative humidity 93%); *patulin*, in a concentration of $10^{-3}M$, administered as the wilt poison. *Abscissae:* hours from start of experiment (indicated by arrow). *Ordinates: w*, fluid intake; *t*, cuticular and stomatal transpiration of the whole shoot, both in g per hour; *f*, fresh weight of shoot in g. At 1, the shoot is limp and its extremities wilt (After Gäumann and Jaag (16)).

such; it is only an accompaniment, though it indicates that toxigenic impairment of the protoplasts has set in in the shoots concerned.

With increasing accumulation of toxin in the leaf or other tissues, the concentration gradually surpasses the critical threshold for impairment of the semipermeability of the boundary layers of the plasma (Table 1). Because of this, the osmotic conditions for turgor are destroyed, and in consequence an irreversible toxigenic wilting sets in in the diseased shoots. Thus, this type of wilting bears no direct relation to the water economy of the diseased shoot, since it also occurs in a moisture-saturated atmosphere in which the plants cannot lose water (Fig. 8B).

Loss of turgor following reduction of water supply.—In diseased tomato shoots, water transport may be reduced to about 2%; if the transpiration stream in healthy tomato plants is advancing 0.55 cm/second, the rate of ascent in moderately diseased shoots will fall to 0.02 cm/second, and in severely diseased shoots to 0.01 cm/second (11).

Three types of mechanism cooperate in this reduction of the transpiration stream: 1) a toxigenic impairment of the metabolic processes that provide the conditions for ascent of the transpiration stream; 2) a mechanical occlusion of the lumen of the vessels; and 3) a mechanical occlusion of the intermicellar spaces of the cell walls.

1) *Toxigenic impairments of the metabolic conditions for ascent of the transpiration stream.*—Toxigenic impairment of the protoplasts significant for the transpiration stream occurs in the root system of the diseased plants as soon as infection finds its way into the roots. Even in the first days of infection, *Fusarium lycopersici* and *Fusarium vasinfectum* form considerable amounts of fusaric acid; hence, the ground tissue cells affected by this acid in the neighborhood of the focus become "turgor sick," and their metabolism is impaired, leading to a reduction of root pressure.

Further, the vascular bundles in the shoots of the diseased plants suffer toxigenic injuries, which later manifest themselves in the familiar necroses and postmortem brownings. Plant physiologists have provided irrefutable evidence that the vessels contribute nothing to the ascent of the transpiration stream, and that this is forced or sucked quite passively through them without their aid. On the other hand, the xylem parenchyma helps at least by maintaining the suction pressure in the vessels (50). Thus, if the parenchyma becomes diseased, the efficiency of the vessels is presumably lowered.

Finally, the protoplasts of the leaves also are toxigenically injured (57), so that their suction power and capacity to replace the transpired water become defective.

2) *Occlusion of the vessels as a cause of reduction in water supply.*—In the fusarioses to be discussed here, the occlusion of vessels by the hyphae of the pathogen or by amorphous pectin gels (11, 22, 24, 42, 49, 53, 60) occurs mainly in the stems. Under certain experimental conditions an average of 2.59 ml of water per hour passes through a 30-cm length of stem of a healthy tomato plant, whereas only 0.13 ml passes through a similar length of stem of a diseased plant (only about 5% of the former amount). Forcing a given quantity of fluid through the stem in a unit of time requires a pressure of about 0.1 atmosphere for healthy shoots, and about 2 atmospheres for diseased shoots (42).

However, it is difficult to estimate the value of these mechanical hindrances to the transpiration stream within the living plant; in the tomato shoot the regulatory and compensatory powers are so great that the shoot does not wilt even when 83% of its vessels are put out of action (42).

3) *Mechanical occlusion of the intermicellar spaces of the cell walls as a cause of reduction in water supply.*—The transpiration stream that ascends through the stem in the lumen of the vessels passes through the vessel walls into the cell walls of the ground tissues, in this way bathing the protoplasts on all sides. In wilt-sick tomato shoots the transpiration

stream carries with it foreign bodies of various sizes, some dissolved off the cell walls and others formed by the pathogen itself.

The *host* particles derive mainly from pectin molecules that have become soluble and have been fragmented by the pathogen's depolymerases (22). How far the toxins of the pathogen—fusaric acid in particular—prepare the way for these events has not yet been ascertained.

The particles contributed by the *pathogen* consist mainly of polysaccharide slimes resulting from hyphal autolysis. In the case of *F. lycopersici* they probably play only a minor role since (at least *in vitro*) they are not formed in larger quantities before about the third week (11). However, in certain other wilt pathogens—*Fusarium solani* (Mart.) App. et Wr. (58) and *Bacterium solanacearum* E. F. S. (39), for example—slime is produced in pathogenic amounts.

Because of their size, these particles cannot pass with the transpiration stream through the micellar felt of the cell walls. Hence, they are caught up in it and choke the intermicellar spaces (the submicroscopic capillaries) of the micellar felt, and probably also the intercellular spaces and the terminal vascular ramifications in the leaves, thus interrupting the passage of transpiration water. In this way an irreversible "submicroscopic embolism" is established in the leaves and in the ground tissues of the stem (17, 27, 28, 29, 39, 43).

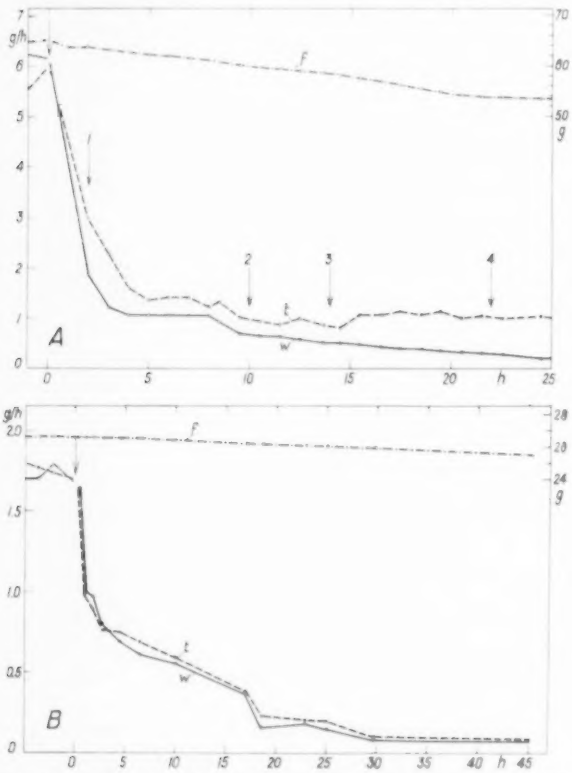


FIG. 9. The impairment of water economy in a tomato shoot—A) by bacterial slimes; B) by a $10^{-3}M$ solution of inulin. *Abscissae:* hours from start of experiment—*w*, water intake; *t*, shoot transpiration (both in mg/hour); *f*, fresh weight (g) of shoot. At 1, the tip droops, the leaves become sleepy; at 2, grooves in the stem; at 3 the stem bends down; at 4, entire plant wilted. (After Kunz (39) and Gäumann and Jaag (17)).

The sites where this occlusion of the intermicellar spaces occurs are determined by the size of the particles.

Large particles (molecular weight some 10,000) are already caught in the walls of the vessels and of their neighboring cells, thereby hindering the passage of water from the vessels into the ground tissues. In Fig. 9A a bacterial slime from slimy wine (molecular weight 50,000-100,000) was selected as a chemically inert substance for a model experiment. After 5 hours the water intake of the shoots amounted to only 15% of the initial value, whereas transpiration remained at 20%; the output was thus about one third greater than the simultaneous intake (39). This disharmony leads to water shortage in the distal tissues, and thereby a loss of turgor, and so to wilting. In this case, as with vessel occlusion, the wilt effect is induced by a local insufficiency of water; but here the blocking of the transpiration stream does not occur in the lumen of the vessels but in the intermicellar spaces of the walls of the vessels and of the adjacent parenchymatous cells.

Smaller particles (molecular weight under 10,000), on the other hand, can travel widely in the cell walls of the parenchymatous tissues, so that the submicroscopic embolism first arises there. In Fig. 9B, inulin (molecular weight c. 5000) was chosen as a model substance. Its molecules block the water routes of the ground tissues so completely that no more water can be absorbed by the cells though none can be surrendered. Their entire water exchange is therefore paralyzed; as a result, the fresh weight of the shoot remains constant, and no obvious injuries appear in it during the first 48 hours (17). Thus, particles with smaller molecules do not give rise to a local deficiency of water, and there is no pathological wilting of the host plants; they simply shut off the affected protoplasts from the transpiration stream. Perhaps an observation by Davis (9)—that, in cloudy weather, wilt-sick tomato plants show guttation, i.e. water congestion—can be explained by the occlusion of the submicroscopic water routes by small particles: "Thus, it would appear that the wilting . . . was due to an inability to utilize the water passing through the stock rather than an impairment of the water transporting capacity of the stock."

Thus, parasitigenic wilting, like other nonspecific syndromes of the wilting diseases—yellowing, brittleness, necroses, discoloration of vessels, etc.—has an exceedingly complex basis. We not only know little about the quantitative and temporal relations in the interplay of these factors, but they may differ from one strain of pathogen to another, and also according to the reactivity of the host.

Hence, there need be no contradiction when Gottlieb (24) speaks of a *reversible* wilting in fusariosis

of tomato, and Thatcher (57) of an *irreversible* wilting in the same subject. In our opinion, different components of the parasitogenic wilting were dominant in the 2 cases. In Gottlieb's case the loss of turgor that led to wilting depended mainly on a local water deficiency caused by mechanical occlusion in the vessels and in the intermicellar spaces of the cell walls; in Thatcher's case it depended mainly on direct toxigenic impairment of the semi-permeability of the protoplasts in the wilting tissues.

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INHERITANCE OF RESISTANCE TO ROOT-KNOT NEMATODES IN TOBACCO[1]

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SUMMARY

Nicotiana tabacum plants resistant or susceptible to root knot caused by *Meloidogyne incognita* var. *acrita* were crossed in various combinations as a part of a program of breeding for disease resistance. F_1, F_2, F_3, and backcross progenies were grown in root-knot-infested soil in greenhouse and field. Their response to root knot in both seedling and mature stages support the hypothesis that a single dominant factor, or a block behaving as a single factor, controls resistance to the *M. incognita* var. *acrita* populations used in this study. However, in certain parental combinations, excesses of either resistant or susceptible plants occurred.

Resistance to root-knot nematodes, *Meloidogyne* spp., has been found in a number of economic plants. Genetic studies of resistance show that inheritance patterns vary from simple to complex. Barrons (2) reported that 2 recessive genes controlled resistance in garden beans. Studies in tomato (6) suggested that resistance to *M. incognita* (Kofoid & White, 1919) Chitwood, 1949, was dominant and conditioned by 1 major gene. Another investigation in tomato (1) indicated that resistance to 4 *Meloidogyne* spp. was governed by 1 incompletely dominant gene. Hare (8) reported that a single dominant gene in pepper controlled resistance to 2 species of root knot. Studies in cotton (11) suggest a complex inheritance of resistance. Reports concerning peach (13) and grape (9) were inconclusive since only F_1 data were presented. Data from F_2 families of lespedeza (7) gave high heritability values for resistance to 2 forms of root knot, with resistant, intermediate, and susceptible types present.

Resistance to root knot in *Nicotiana tabacum* L. was reported by Clayton and Foster (3) in 1940. A summary (4) has been prepared on procedures used to incorporate this resistance into flue-cured breeding lines, and data have been presented for F_2 populations indicating that resistance is controlled by a single dominant gene, but with modifying factors involved.

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Information about the manner of inheritance of characters is valuable for conducting a plant improvement project. The results presented here were obtained as a part of a program in breeding for disease resistance in flue-cured tobacco. This report on inheritance of resistance to root knot includes data obtained from seedling tests in the greenhouse and from mature plants tested in the field. A preliminary report (5) has been given.

MATERIALS AND METHODS.—The source of root-knot resistance used in this study was derived from TI 706[2] as reported by Clayton et al (4) and obtained from the Tobacco Section, U. S. Department of Agriculture. Susceptible parents included the flue-cured varieties Dixie Bright 101, Hicks, and Coker 139, as well as breeding lines. Crosses were made in the field and greenhouse with adequate precautions to avoid contamination with foreign pollen. Seed used in all progeny tests came from inflorescences that had been bagged to prevent cross-pollination.

Heterogeneity chi-square values were calculated whenever progenies of similar origin were combined (12). This method was used to determine whether sample ratios were homogeneous.

Greenhouse.—Seedlings were germinated and grown in steam-sterilized soil for 4–5 weeks, then transplanted, either individually into 2-in. clay pots or in sets of 4 into 4-in. pots, in soil heavily infested with *M. incognita* var. *acrita*. After a minimum of 4 weeks the roots were washed in water and examined for galling and reproduction of the nematode. Plants were divided into 2 classes: 1) resistant, with clean roots; or 2) susceptible, with galled roots. All tests included susceptible check plants.

Field.—Progenies were seeded in plant beds and transplanted to fields naturally infested with *M. incognita* var. *acrita* in North Carolina or South Carolina during 1955–57. Two or 3 replicates, of 15–25 plants each, were grown for approximately 120 days. Roots were then examined, and classified as in the greenhouse studies. Inheritance data were obtained only where susceptible check plants were galled. This reduced the total number of plants in certain progenies since root knot was not uniformly distributed within fields.

RESULTS.—The results obtained with 5 groups of tobacco seedlings grown in root-knot-nematode-infested soil under greenhouse conditions are presented in Table 1. Groups A, B, and C, representing 3 crosses of resistant × susceptible plants, show the parent and progeny reaction to *M. incognita* var. *acrita*. In all 3 crosses the behavior of the parental material was as expected. Resistance as shown by absence of galled plants was completely dominant in the F_1 of group A. However, the F_1 progenies of groups B and C contained, respectively, 5 and 2 galled plants. Nevertheless, the F_2 populations in the 2 groups agreed satisfactorily with the hypothesis that a single dominant factor controls resistance to *M. incognita* var. *acrita*. Whereas there was an excess of resistant plants in the F_2 of Group A, backcrosses to both parents in groups A and B fit the expected ratios for a single dominant factor. In backcrosses to resistance the parents used were similar to those in the original crosses, but not identical. In group A it is assumed that the backcross parent substituted was heterozygous for root-knot resistance. However, the probability value was low for the expected 3:1 resistant-susceptible ratio. The F_1 seedlings in group D originated from crosses between resistant and susceptible plants in 1957. Of 556 plants inoculated, 3 developed galls, emphasizing the dominance of resistance with various susceptible parents. Results from the 22 segregating F_3 lines in group E agreed well with the expected 3:1 ratio. These were part of a test of 28 F_3 progenies where 6 were homozygous resistant and 22 segregated. The P value was 0.10–0.20 for the expected 1:2 homozygous-heterozygous among the 28 resistant F_2 parent plants.

Data obtained from F_1 plants in the field and seedling F_2 progenies in the greenhouse are shown in Table 2. F_1 numbers 103, 105, and 106 originated from crosses of heterozygous resistant × susceptible parents, and agreement was satisfactory for the expected 1:1 backcross ratio. F_2 populations originating within each of these crosses were combined. An

TABLE 1.—*Seedling reaction of resistant and susceptible parents and progenies of tobacco when grown in root-knot-nematode-infested soil*

	Parent or progeny	Observed no. R[a]	Observed no. S[a]	Expected ratio[a]	P[b]
A	P_1	67	0	R	
	P_2	0	84	S	
	F_1	108	0	R	
	F_2	189	44	3:1	0.02–0.05
	P_2 x F_1	71	68	1:1	0.70–0.80
	F_1 x P_2	29	24	1:1	0.30–0.50
	P_1[c] x F_1	192	46	3:1	0.02–0.05
B	P_1	22	0	R	
	P_2	0	64	S	
	F_1	67	5	R	
	F_2	145	38	3:1	0.10–0.20
	F_1 x P_2	96	76	1:1	0.10–0.20
	P_1[d] x F_1	29	0	R	
C	P_1	24	0	R	
	P_2	0	28	S	
	F_1	56	2	R	
	F_2	113	47	3:1	0.20–0.30
D	30 F_1 progenies	553	3	R	
	S check	0	74	S	
E	22 segregating F_3 lines	659	213	3:1	0.50–0.70
	S check	0	169	S	

[a] R = resistant; S = susceptible. Ratios expressed as R:S.
[b] Probability value from chi-square test for goodness of fit.
[c] Bel 5-47.
[d] Bel 5-48.

[2] Accession number of the Tobacco Section, U. S. Department of Agriculture, and refers to Plant Introduction 116,080.

TABLE 2.—*Reaction of F_1 plants in the field and seedling F_2 progenies in the greenhouse, each grown in root-knot-nematode-infested soil*

	F_1			F_2[a]			
	Observed no.				Observed no.		
Field no.	R[b]	S[b]	Expected ratio	No. of progenies	R[b]	S[b]	P[c]
103	22	18	1:1	7	97	22	0.10–0.20
105	20	17	1:1	6	90	16	0.01–0.02
106	13	20	1:1	8	100	44	0.10–0.20
107 etc.[d]	217	0	Resistant	52	801	201	<0.01
S check	15	105			0	126	

[a] About ½ of F_2 plants grew to maturity in root-knot-nematode infested soil.
[b] R = resistant; S = susceptible.
[c] Probability value from chi-square test for goodness of fit for 3:1 ratio.
[d] Seven F_1 progeny (107–110, 112, 119, 120).

excess of resistant plants occurred in the 105 group, and chi-square interaction for the 106 group was highly significant, suggesting that the samples were heterogeneous. Two F_2 populations from 106, with respective counts of 6 and 7 resistant and 12 and 11 susceptible, were presumed responsible for the deviation.

Root-knot data from 7 F_1 populations of homozygous resistant × susceptible parents and their respective F_2 progenies, shown in Table 2, indicate complete dominance and monofactorial inheritance of resistance. However, F_2 progenies contained an excess of resistant plants.

The root-knot reaction of mature plants of segregating progenies is presented in Table 3 and illustrated in Fig. 1. Agreement with expected ratios was generally satisfactory for the hypothesis that a single dominant factor governs resistance. There was a surplus of susceptible plants in the 24 F_2 progenies derived from the first backcross to susceptible parents. Two of the 24 progenies had P values less than 0.05. However, plants grown in 1957 from remnant seed of these gave good agreement with the expected ratio. Also, in 1957, 47 F_3 progenies from resistant F_2 plants were grown in the field nursery. Fifteen lines were homozygous resistant and 32 segregated, giving a P value of 0.80–0.90 for the expected 1:2 homozygous-heterozygous ratio. Individual plant counts in 14 of the 32 segregating lines showed an excess of resistant plants (Table 3), with the same tendency in 15 F_2 populations from crosses of susceptible × resistant parents.

DISCUSSION.—In general, data from both seedlings and mature plants support the hypothesis that a single dominant factor, or a block behaving as a single factor, governs resistance to the populations of *M. incognita* var. *acrita* from North and South Carolina used in this study. Deviations from expected ratios were noted, with resistant plants in excess whenever probability values for progeny groups were less than 0.01. Since all susceptible check plants in greenhouse studies were galled, the probability of escapes among resistant plants is low. Hence, modifying gene(s) augmenting resistance appear to be present in some parental combinations. Clayton et al (4) emphasized that the action of modifiers was evident with certain parents. Certain progeny groups, however, had excesses of susceptible plants, with the greatest deviations in small F_2 populations of F_1 106 (Table 2). This progeny is of special interest because a surplus of susceptible plants occurred in the F_1 and F_2 generations, indicating that factor(s) enhancing resistance were perhaps absent. The chance that this will occur often seems to be small when considering the relatively large number of parental combinations involved in this study. Possible explanations for the occurrence of susceptible F_1 plants may be contaminated pollen, a seed mixture, or the

TABLE 3.—*Mature plant reaction of segregating progenies grown in fields naturally infested with root-knot nematodes*

Year	No. of progenies	Origin[a]	Generation	Observed no. R[a]	Observed no. S[a]	Expected ratio	P[b]
1955	11	R × S	F_2	444	127	3:1	0.10–0.20
		S check		2	190		
1956	7	2nd Bc to S	F_1	242	249	1:1	0.70–0.80
	3	F_1 × F_1	F_1	159	52	3:1	0.90–0.95
	24	1st Bc to S	F_2	547	213	3:1	0.05–0.10
	8	R × S	F_2	127	39	3:1	0.50–0.70
		S check		7	218		
1957	13	2nd Bc to S	F_2	258	99	3:1	0.20–0.30
	15	S × R	F_2	296	82	3:1	0.10–0.20
	14	segregating lines	F_3	288	56	3:1	<0.01
		S check		0	363		

[a] R = resistant; S = susceptible; Bc = backcross.
[b] Probability value from chi-square test for goodness of fit.

FIG. 1. Roots of resistant and susceptible tobacco plants from an F_2 population grown in soil infested with root-knot nematode.

presence of modifiers in certain combinations.

Plants with the same source of resistance, grown in the greenhouse, were highly resistant to *M. incognita*. Variation in pathogenicity has been reported in isolates of *M. incognita* and *M. incognita* var. *acrita* (10), indicating that a resistant line may be susceptible under some conditions. This parallels situations where varieties resistant to other diseases are planted widely and are later reported as susceptible in certain localities. Consequently, it is probable that the continued usefulness of root-knot-resistant varieties will depend on the development of additional sources of resistance.

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CHEMICAL CONTROL OF CEREAL RUSTS. III. THE INFLUENCE OF NICKEL COMPOUNDS ON RYE LEAF RUST IN THE GREENHOUSE[1]

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SUMMARY

Nickel compounds applied as protective sprays to rye plants in 4-in. pots showed the following order of effectiveness on *Puccinia rubigo-vera:* acetate = acrylate = carbonate = chloride = fluoride = fluoborate > bromide = citrate = nitrate = sulfate = sulfide > cyanide > hydroxide. The same compounds applied as eradicative sprays 5 days after inoculation (fleck stage) showed the following order of effectiveness: acetate = acrylate = bromide = chloride = fluoborate = fluoride = nitrate = sulfate = sulfide > carbonate = citrate > hydroxide > cyanide. All but the fluoborate showed little or no phytotoxicity at the level of 0.5 lb. per 100 gal. Bis[*N*-(2-hydroxyethyl)dodecylmethylbenzylamine] nickel (II) chloride (RH-1) and *N*-(2-hydroxyethyl) dodecylmethylbenzylamine (RH-8) in simulated "rain" tests showed little loss of excellent protective control; nickel chloride hexahydrate (RH-9) lost poor protective control completely. Both RH-1 and RH-9 gave very good eradication despite "rain"; RH-8 was essentially ineffective. Nickel chloride hexahydrate was about 3 times as effective as an eradicant than as a protectant. The time when rye leaf rust appears most vulnerable to eradication with the nickel compounds is the fleck stage (4–6 days after inoculation). Spray applications preceding or following this stage give less eradication of the rust. When the rust is treated (fleck stage) by spraying with water-soluble nickel chloride hexahydrate and then exposing the plants to "rain" before drying, only a few minutes of exposure appeared necessary for effective eradication. Formulations of nickel chloride hexahydrate and Dithane gave very effective protection and eradication even when exposed to rain.

Papers I and II (4, 6) of this series showed that certain nickel amine complexes control cereal rusts. Paper I reported that the eradicative property appeared due to the nickel part of the molecule, and that the protectant property was associated with both the amine and nickel parts. Paper II reported that certain nickel amine complexes satisfactorily controlled wheat rust under field conditions. The present study was therefore undertaken to evaluate and compare the nickel amine complexes with cheaper and more accessible organic and inorganic nickel compounds.

MATERIALS AND METHODS.—The methods used were the same as those described in paper I (4). Briefly, the protective tests were conducted by treating the plants before inoculation, and the eradicative tests by treating 5 days after inoculation (fleck stage). Urediospores of *Puccinia rubigo-vera* (DC.) Wint. f. sp. *secalis* (Ericks.) Carleton collected from greenhouse-grown Balbo rye plants were used in all studies. (The binomial *Puccinia recondita* Rob. ex Desm. has recently been suggested for this fungus (1), by Cummins and Caldwell.) Spore suspensions (25,000 per ml) contained: 0.1% sucrose and 0.001% potassium citrate, to aid germination, and 0.0025% Triton NE (an alkyl aryl polyether alcohol, Rohm & Haas Co.), to improve spore dispersion and wetting of the foliage. Fifty 10–12-day-old Balbo rye seedlings per 4-in. pot, trimmed to a height of 4.5 in. and sprayed with 7 ml of spore suspension, showed 1500–1800 pustules.

In both protective and eradicative tests the chemical was sprayed on the plants to the point of run-off, requiring about 20 ml per pot. To aid wetting and spreading, either Triton B-1956 (a modified alkyd resin, Rohm & Haas Co.) or Triton X-114 (an alkyl aryl polyether alcohol, Rohm & Haas Co.) at 1:4000 was used with all chemical compounds. After inoculation, incubation, and treatment, the plants were placed on the greenhouse bench until evaluation for rust. In protective tests the plants were held 11 days in the greenhouse after 24 hours of incubation in a humidity cabinet (20°C and 100% relative humidity); in eradicative tests they were held only 7 days in the greenhouse after treatment at the fleck stage.

Simulated rain tests were conducted by placing treated dry plants on a rotating turntable and spraying them with tap water through a Teejet nozzle #8003E: 2 in. of "rain" in about 20 minutes. In protective tests the plants were treated with the chemical and exposed to the "rain" before inoculation and incubation; in eradicative studies the order was inoculation, incubation, treatment with chemical, and "rain." In all cases, unless otherwise specified, "rain" was not applied until the chemical had dried on the plants.

In both protective and eradicative tests, rust development was recorded by indexing each pot of 50 plants as follows: 0 = no pustules; 0.25 = 1–5 pustules; 0.5 = 6–20 pustules; 0.75 = 21–50 pustules; 1.0 = 51–100 pustules; 2 = 101–200 pustules; 3 = 201–500 pustules; 4 = 501–1000 pustules; and 5.0 = >1000 pustules.

RESULTS.—Preliminary studies with nickel amine complexes showed that the protective property of these compounds is associated with both the amine and nickel parts of the molecule, and that the eradicative property is associated with the nickel. This was strikingly brought out in simulated rain studies, reported in Table 1. Bis[*N*-(2-hydroxyethyl)dodecylmethylben-

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TABLE 1.—*Effects of simulated rain (2 in.) on protective and eradicative control by bis N-(2-hydroxyethyl) dodecylmethylbenzylamine nickel (II) chloride (RH-1) and its component parts*

Compound[b]	Amt. per 100 gal. water[c]	Rust index[a]				Plant injury
		Protectant		Eradicant		
		No rain	Rain	No rain	Rain	
Bis[*N*-(2-hydroxyethyl) dodecylmethylbenzylamine] nickel II chloride (RH-1)	2 lb.	0.0	0.0	0.0	0.0	Moderate
	1 lb.	0.0	0.6	0.1	0.6	Slight
	0.5 lb.	0.25	1.0	0.6	2.0	Very Slight
N-(2-hydroxyethyl) dodecylmethylbenzylamine (RH-8)	760 g	0.0	0.0	1.0	3.0	Moderate to severe
	380 g	0.25	0.25	5.0	5.0	Slight
	190 g	0.5	0.6	5.0	5.0	Very slight
Nickel chloride hexahydrate (RH-9)	272 g	0.75	4.5	0.0	0.0	Slight
	136 g	1.5	5.0	0.4	0.5	None
	68 g	3.0	5.0	0.6	0.9	None
Untreated		5.0	5.0	5.0	5.0	

[a] Based on average of 2 50-plant pots: 0 = no pustules; 0.25 = 1–5 pustules per 50 plants; 0.5 = 6–20; 0.75 = 21–50; 1.0 = 51–100; 2.0 = 10–200; 3= 201–500; 4 = 501–1000; 5 = >1000.
[b] Triton X-114 (alkyl aryl polyether alcohol) at rate of 1:4000 added to aid wetting and spreading.
[c] There are 380 g of the amine RH-8 and 136 g of RH-9 in every lb. of RH-1.

zylamine] nickel (II) chloride (RH-1) and *N*-(2-hydroxyethyl) dodecylmethylbenzylamine (RH-8), from which the first is made, showed excellent protective control and withstood 2 in. of "rain" with very little loss of effectiveness. The nickel chloride hexahydrate (RH-9), however, showed poor to fair protective control, which was lost almost completely upon weathering. The eradicative test demonstrated that both RH-1 and RH-9 gave very good control, with or without exposing them to "rain," whereas the free amine (RH-8) was essentially ineffective. It was also noted that both RH-1 and RH-8 were more phytotoxic than was RH-9.

Control with simple organic and inorganic nickel compounds.—Table 2 presents typical data obtained with simple organic and inorganic nickel compounds in protective or eradicative tests, with and without exposure to "rain." Quite obviously, most of the compounds tested are very good protectants. In fact, all show a reduction in the number of pustules, with nickel hydroxide showing the least. It is further noted that, in the protective tests, practically all the compounds, with a few exceptions, become almost completely ineffective when subjected to "rain." The 2 compounds least affected by "rain" are the fluoride and fluoborate. From protective tests without exposure to "rain," the nickel compounds may be arranged in the following order of effectiveness: chloride = acetate = acrylate = carbonate = fluoride = fluoborate > citrate = nitrate = sulfate = sulfide = bromide > cyanide > hydroxide.

All the compounds tested are very effective eradicants. From eradicative studies without exposure to "rain" the compounds may be arranged in the following order of effectiveness: acetate = acrylate = bromide = chloride = fluoborate = fluoride = nitrate = sulfate = sulfide > carbonate = citrate > hydroxide > cyanide. It is noteworthy that all the compounds except nickel fluoborate produced little or no plant injury.

The eradicative action of the chemicals was not destroyed by "rain," although "rain" washes off practically all of the compounds, making them ineffective for protection. The results indicate that the chemical

TABLE 2.—*Effect of simulated rain (2 in.) on protective and eradicative rust control with various nickel compounds*

Compound[b]	Lb. per 100 gal.[c]	Rust index[a]			
		Protectant		Eradicant	
		No rain	Rain	No rain	Rain
Untreated		5.0	5.0	5.0	5.0
Nickel chloride hexahydrate	0.5	0.5	4.0	0.0	0.13
Nickel chloride hexahydrate	0.25	0.9	5.0	0.4	0.75
Nickel acetate	0.5	0.6	3.5	0.13	0.25
Nickel acetate	0.25	0.9	5.0	0.5	0.9
Nickel acrylate	0.5	0.5	4.0	0.0	0.4
Nickel acrylate	0.25	0.9	5.0	0.6	0.9
Nickel carbonate	0.5	0.5	5.0	0.5	0.5
Nickel carbonate	0.25	0.9	5.0	0.9	1.0
Nickel citrate	0.5	0.6	5.0	0.25	0.9
Nickel citrate	0.25	1.5	5.0	1.0	2.0
Nickel cyanide	0.5	1.0	5.0	2.5	3.5
Nickel cyanide	0.25	2.5	5.0	5.0	5.0
Nickel fluoride	0.5	0.5	2.0	0.0	0.4
Nickel fluoride	0.25	0.75	4.0	0.25	0.5
Nickel fluoborate	0.5	0.25[d]	1.0[d]	0.0	0.0
Nickel fluoborate	0.25	0.5[e]	2.0[e]	0.0	0.25
Nickel hydroxide	0.5	3.0	5.0	0.5	3.5
Nickel hydroxide	0.25	5.0	5.0	1.5	5.0
Nickel nitrate	0.5	0.75	5.0	0.0	0.13
Nickel nitrate	0.25	1.5	5.0	0.4	0.5
Nickel sulfate	0.5	0.9	5.0	0.13	0.5
Nickel sulfate	0.25	1.5	5.0	0.4	0.75
Nickel sulfide	0.5	0.75	5.0	0.4	0.5
Nickel sulfide	0.25	1.0	5.0	0.5	0.9
Nickel bromide	0.5	0.5	5.0	0.0	0.4
Nickel bromide	0.25	1.0	5.0	0.4	0.6

[a] See footnote *a* in Table 1 for scale of index.
[b] Triton B-1956 (modified alkyd resin, Rohm & Haas Co.) at rate of 1:4000 added to aid spreading.
[c] Applied 20 ml of this concentration to each 4-in. pot of 50 rye plants 4.5 in. tall; each treatment replicated twice.
[d] 1 lb. per 100 gal.
[e] ½ lb. per 100 gal.

TABLE 3.—*Eradicative control*[a] *as influenced by interval between treatment*[b] *with nickel chloride hexahydrate and exposure to simulated rain (2 in.)*

Compound[c]	No rain	1 minute	5 minutes	15 minutes	30 minutes	1 hour	2 hours	4 hours
RH-9 0.5 lb./100 gal.	0.0	2.5	0.75	0.5	0.25	0.0	0.0	0.0
Untreated	5.0							

[a] See footnote *a* in Table 1 for scale of index.
[b] At time of treatment, the rust was in the fleck stage, with about 25% of the sori showing signs of rupture.
[c] Triton B-1956 (modified alkyd resin, Rohm & Haas Co.) at rate of 1:4000 added to aid wetting and spreading.

in eradicative tests is very rapidly picked up by the fungus, at least within the 2-hour period normally allowed for the treatments to dry.

Influence on eradication of interval before "rain."—It was assumed that water-soluble nickel chloride hexahydrate would be washed off by "rain" in the eradicative test because previous protective studies showed this to be true. A test was designed to determine the minimum period of exposure to the chemical necessary for effective eradication of rust. The results are presented in Table 3. It should be remembered that the plants were treated at the fleck stage, with about 25% of the sori showing signs of rupture. The plants were kept in the greenhouse at 22°C during the drying period. The plants were still quite wet after 1, 5, and 15 minutes of drying; some droplets of spray material still remained after 30 minutes of drying, and the plants appeared dry after 1 hour.

Even a 1-minute exposure to nickel chloride hexahydrate (0.5 lb. per 100 gal. water) significantly reduced the number of pustules from over 20 per plant on untreated check plants to 3–7; 5 minutes reduced the number to about 1; 15- and 30-minute exposures reduced the number to a fraction of 1; exposures over 1 hour completely prevented rust development.

Influence on protective and eradicative control of amount of nickel.—It was next desirable to determine the quantities of nickel necessary for satisfactory control. Data in Table 4 show that nickel chloride hexahydrate caused some eradication at 25 g (6.2 g nickel) per 100 gal. but was significantly more effective at the 50-g level (12.4 g nickel). In fact, at the 50-g level, pustule development was less than 10% of that of the untreated checks. These data indicate that the breakpoint for nickel chloride hexahydrate as an eradicant is between 25 and 50 g per 100 gal. when applied at the rate of 20 ml per 4-in. pot of 50 plants. As a protectant the breakpoint appears between 70 (17.3 g nickel) and 140 g (34.6 g nickel) per 100 gal. These data apparently indicate that nickel chloride hexahydrate is a somewhat poorer protectant than eradicant.

Table 4 also indicates that nickel chloride hexahydrate is a relatively safe compound on rye. In fact, the margin of safety on this crop seems quite wide: objectionable injury usually does not appear with less than 1.5 lb. per 100 gal., and it is conceivable that injury in the field might be far less when the plants

TABLE 4.—*Protective and eradicative control of rye leaf rust with nickel chloride hexahydrate*

Concentration[a] (g)	Nickel (g)	Rust index[b] Protectant	Eradicant	Plant injury
804[c]	198.6	0.1	0.0	Moderate
704[c]	174.0	0.1	0.0	Moderate to severe
402[c]	99.3	0.5	0.0	Slight
280[c]	69.2	0.5	0.3	Slight to Moderate
201[d]	49.5	0.75	0.1	Very slight
140[d]	34.6	0.9	0.1	Very slight
70[d]	17.3	2.5	0.4	None
50[d]	12.4	2.8	0.8	None
25[d]	6.2	4.1	3.3	None
12[d]	3.0	5.0	5.0	None
Untreated		5.0	5.0	None

[a] Active ingredient per 100 gal. of spray.
[b] See footnote *a* in Table 1 for scale of index.
[c] Triton B-1956 (modified alkyd resin, Rohm & Haas Co.) at rate of 1:4000 added to aid spreading.
[d] Triton X-114 (alkyl aryl polyether alcohol, Rohm & Haas Co.) at rate of 1:4000 added to aid spreading.

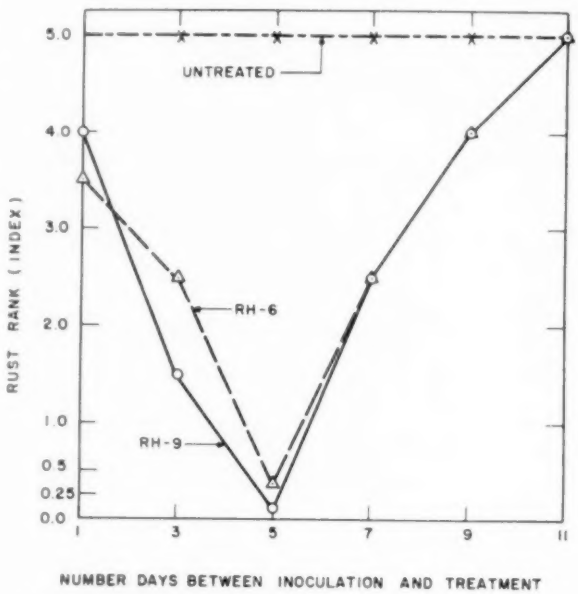

FIG. 1. Influence of application time on rye leaf rust eradication. Based on average of 2 50-plant pots: 0 = no healthy pustules, 0.25 = 1–5 pustules per 50 plants; 0.5 = 6–20; 0.75 = 21–50; 1.0 = 51–100; 2.0 = 101–200; 3.0 = 201–500; 4.0 = 501–1000; 5.0 = >1000. Note that equal quantities of nickel supplied in the form of bis[*N*-(2-hydroxyethyl) dodecylbenzylamine] nickel (II) chloride (RH-6) and nickel chloride hexahydrate (RH-9) are used in all applications.

TABLE 5.—*Eradication of rye rust with zineb (Dithane Z-78) plus nickel chloride hexahydrate (RH-9)*

Treatment[a]	Amt./100 gal.	Rust index[b]
Untreated		5.0
Dithane Z-78	0.5 lb.	5.0
RH-9	201 g	0.1
RH-9	150 g	0.1
RH-9	100 g	0.4
RH-9	50 g	0.8
RH-9	25 g	3.3
RH-9	12.5 g	5.0
Dithane Z-78 + RH-9	0.5 lb. + 201 g	0.1
Dithane Z-78 + RH-9	0.5 lb. + 150 g	0.2
Dithane Z-78 + RH-9	0.5 lb. + 100 g	0.3
Dithane Z-78 + RH-9	0.5 lb. + 50 g	0.8
Dithane Z-78 + RH-9	0.5 lb. + 25 g	3.0
Dithane Z-78 + RH-9	0.5 lb. + 12.5 g	5.0

[a] Triton X-114 (alkyl aryl polyether alcohol, Rohm & Haas Co.) at rate of 1:4000 added to aid spreading.
[b] See footnote *a* in Table 1 for scale of index.

are not as succulent and tender as those grown in the greenhouse.

Influence on rust eradication of application time.—It has been demonstrated in many tests that nickel effectively eradicates rust when applied 5 days after inoculation at the fleck stage. How well would these same compounds eradicate rust if applied at other intervals after inoculation? Test data in Fig. 1 provide an answer. When applied immediately after inoculation, essentially equal eradication is shown by equal quantities of nickel supplied in the form of bis-[*N*-(2-hydroxyethyl)dodecylbenzylamine] nickel II chloride (RH-6) and nickel chloride hexahydrate (RH-9). Maximum eradication is obtained with application 5 days after inoculation.

Combination of nickel with known rust protective fungicides.—As pointed out above, practically all of the cheap organic and inorganic nickel compounds failed to resist "rain" satisfactorily when used as protectants. An attempt was made to overcome this weakness by combining nickel with zineb (Dithane Z-78), which has been proven to be a good protectant in the control of cereal rust. A combination in such a manner might provide both a protectant and eradicant that would weather well. Table 5 presents the results of one such study—on Dithane Z-78 (zinc ethylenebisdithiocarbamate) combined with nickel chloride hexahydrate.

The data show no evidence of eradication of rye leaf rust with Dithane Z-78 alone, but the combination of nickel chloride hexahydrate (RH-9) and Dithane Z-78 gave excellent eradication. When results obtained with RH-9 alone and those from tank-mix Dithane Z-78 plus RH-9 are compared, it is obvious that the eradicative effectiveness depends strictly on the quantity of nickel chloride hexahydrate present, which seems completely compatible with the Dithane.

Protective tests with "rain" showed that Dithane Z-78 resists wash-off, whereas nickel chloride hexahydrate alone is completely lost. These results, with those of the eradicative studies, indicate that a combination of Dithane Z-78 and nickel chloride hexahydrate will provide both protection and eradication that should, under field conditions, resist wash-off by rain or dew.

Combinations of nickel chloride hexahydrate with other Dithanes (M-22, maneb, and tank-mix zineb) also provide good protection and eradication that weather well. When the amount of nickel chloride hexahydrate used is exactly enough to react all the Dithane D-14 in preparation of tank-mix nickel Dithane, protection and eradication result. If a slight surplus of nickel is used, the eradicative property appears to be significantly increased. The margin of safety is much narrower for nickel Dithane than for Dithane Z-78 or M-22.

Some promise of improvement in the sticking properties of nickel chloride hexahydrate has been made by combining it with ammonium hydroxide. Quite striking resistance was shown in preliminary protective tests by preparations of one mole of $NiCl_2 \cdot 6H_2O$ with 2 or 4 moles of NH_4OH (15.1*N*). Eradicative tests showed similar results.

DISCUSSION.—If a fungicide provides both protection and eradication of cereal rust, it is reasoned that such a material should have a chance of becoming a commercial product providing it is cheap and will return to the farmer his investment plus something extra. Whether or not the nickel compounds become commercial products will depend to a certain extent on the efficacy of the cheaper simple organic and inorganic nickel compounds under field conditions.

It is conceivable that there are situations where a material with only the eradicative property would successfully control cereal rust. It is our opinion, however, that if the same compound also possessed the protective property it would be more efficient and used more extensively. To overcome the failure of cheap nickel compounds to resist wash-off during rainy periods, they might be formulated or combined with commercial fungicides known to be good cereal rust protectants that resist weathering. Such a combination of Dithane Z-78 and nickel chloride hexahydrate was shown to be very effective in both greenhouse and field (unpublished). Preliminary tests have also shown that certain stickers added to the water-soluble nickel compounds improve their tenacity somewhat. The results are sufficiently promising to make us hopeful that the right sticker for the desired effect may be found.

It was pointed out above that the nickel compounds give best eradication of rye leaf rust when applied during the fleck stage: apparently 4–6 days after inoculation under optimum conditions. This narrow range would be a decided disadvantage in the field, where one might expect all stages of pustule development at the time of application. These results probably indicate that the fleck stage is the most vulnerable period in the development of the rust. From available data it also seems that eradication is more effective before the fleck stage than after. This is reasonable because, if the rust is allowed to develop

to the point where it is producing an abundance of spores, then hardly enough compound is available to overcome these spores. However, greenhouse studies show that eradication results if these brown spores are treated with high concentrations, in applications repeated a few days apart.

The rupture of the leaf epidermis during sporulation of the pathogen would be expected to facilitate penetration by nickel and play a part in selective accumulation in some cases. It would not, however, explain eradication in those cases where rupture has not yet taken place. These results seem to suggest that the permeability of the epidermal cells immediately above the sorus, at least during the fleck stage, has been changed, thus allowing the nickel to pass through more easily. Microscopic examination of free-hand sections taken during the fleck stage shows the presence of immature spores also. Because of the high rate of respiration and metabolism going on in the sorus at this time, it is expected that these spores and mycelium would absorb large quantities of nickel if brought into contact with it.

Suchorukov (10) reported that he was able to reduce infection of wheat plants by *Puccinia persistens* Plowr. and *P. triticina* Ericks. if the plants were sprinkled with 0.5%, 1% and 3% solutions of pure monoammonium phosphate or urea before inoculation. Preliminary studies with tank-mix combinations of water-soluble nickel chloride hexahydrate with 2 and 4 moles of ammonium hydroxide (15.1N) show striking improvement of rain resistance in protective and eradicative tests. In our studies ammonium hydroxide alone at tested concentrations showed no evidence of either protection or eradication of the rust. Forsyth and Peturson (2), in similar tests on wheat rust under greenhouse conditions, found that the combination of nickel chloride hexahydrate with ammonium hydroxide was almost without effect in eradicative studies. Further work is needed to explain these discrepancies. It is, however, important to note that small-scale field tests with the combination on wheat and rye (unpublished gave satisfactory rust control.

A cursory review of the data obtained with the simple organic and inorganic nickel compounds leads one to believe that the eradicative property is associated with their water-solubility. However, more careful observation shows that this is not true, for both water-soluble and insoluble compounds are effective eradicants (e.g., compare nickel chloride and nickel sulfide).

Radioactive phosphorus, sulphur, and carbon compounds supplied to infected plants accumulate most heavily in the vicinity of the rust pustule (3, 8, 9, 11). Shaw and Samborski (9), as well as Yarwood and Jacobson (11), obtained evidence indicating that the materials accumulate in the host tissue. No attempt was made in the present study to determine if nickel accumulates in the fungus and/or host tissue, although this would seem worthwhile. Investigations at Boyce Thompson Institute (5) showing absorption of large amounts of variously labeled compounds by spores indicate that the case with the nickel compounds might be somewhat similar.

It is possible that nickel accumulates in the suscept tissues surrounding the rust lesion. If this happens we might explain this action by chelation of the nickel with the accumulated amino acids, sugars, and other substances shown to be there (8, 10). Presuming this to be the case, one might expect killing of the rust fungus by 1) movement of essential water and/or growth substances out of the fungus by osmosis into the surrounding suscept cells, or 2) hindrance to movement of essential substances into the fungus, thus preventing development of rust or movement of nickel into the fungus after chelation with amino acids and sugars, where it accumulates and prevents normal metabolism.

After we had discovered that certain nickel amine complexes control rust by foliar application, our attention was called to an article by Sempio (7). He demonstrated protective control of wheat rust (*Puccinia triticina* Eriks.) by placing plants in nutrient solution containing nickel nitrate for 8–10 days before inoculating the plants. Tests in our laboratory show that rye plants are severely damaged when held in solutions of various nickel compounds at the concentrations required for effective rust control.

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PHYTOPATHOLOGICAL NOTES

Longevity of Some Graminicolous Species of Helminthosporium Under Mineral Oil.[1] R. A. Cappellini and C. M. Haenseler.[2] The technique of preserving fungus cultures submerged under sterile mineral oil has gained wide usage since 1943, when Sherf[3] successfully maintained *Fusarium* and *Alternaria* spp. 6 months (extent of test) by this method. The survival of several corn pathogens, apparently unchanged, for 2 years by this method was reported in 1946 by Wernham,[4] and a year later Buell and Weston[5] reported a similar survival period for a number of different genera. Later, Wernham and Miller[6] and Henderson[7] reported the successful maintenance under mineral oil of a number of fungi for periods up to 4 years.

In 1951 the senior author, during the course of a study on the cultural characteristics of 34 graminicolous species of *Helminthosporium*, placed subcultures of isolates of 32 of these species growing on Difco potato-dextrose agar, under sterile mineral oil. These were sent to the junior author, who sealed them with "Parafilm"[8] and kept them at room temperatures, where they remained undisturbed for 7 years. Transfers from these 7-year-old cultures were made onto Difco potato-dextrose agar with 1% Difco yeast extract added. Sixteen of the 32 species were recovered within 5-10 days after transfer; the remaining showed no growth after 1 month (Table 1). The most significant change observed in the recovered cultures was an apparent loss of sporulation by the *Helminthosporium dictyoides*, *H. gramineum*, and *H. victoreae* isolates. Slight differences were noted in color of the colony of *H. catenarium*, *H. erythrospilum*, *H. triseptatum*, and *H. tritici-repentis*, but these are considered insignificant. None of the isolates were tested for pathogenicity.

Henry[9] reported the successful maintenance of 17

Table 1.—*Recovery of graminicolous species of Helminthosporium and changes observed after 7 years of storage under sterile mineral oil*

Species	Recovery[b]	Changes in cultural characteristics[a]		
		Color	Sporulation[c] Before	After
H. bromi Died.	—		+	
H. buchloës (Ells. & Ev) LeFebvre & A. G. Johns.	+	unchanged	+	+
H. carbonum Race I Ullstrup	—		+	
H. carbonum Race II Ullstrup	+	unchanged	+	+
H. catenarium Drechs.	+	changed	—	—
H. cyclops Drechs.	+	unchanged	+	+
H. cynodontis Marig.	+	unchanged	—	—
H. dictyoides Drechs.	+	unchanged	+	—
H. erythrospilum Drechs.	+	changed	+	+
H. giganteum Heald & Wolf	—		—	
H. gramineum Rabh.	+	unchanged	+	—
H. halodes Drechs.	+	unchanged	—	—
H. leersiae Atk.	—		—	
H. leucostylum Drechs.	—		+	
H. maydis Nisik. & Miy.	—		+	
H. micropus Drechs.	—		+	
H. monoceras Drechs.	—		+	
H. nodulosum (Berk. & Curt.) Saac.	+	unchanged	—	—
H. oryzae Breda deHaan	—		+	
H. ravenelii Berk. & Curt.	—		+	
H. rostratum Drechs.	—		+	
H. sacchari (Breda deHaan) Butl.	—		+	
H. sacchari[d]	+	unchanged	+	+
H. sativum Pam., King & Bakke	—		+	
H. setariae Saw.	—		+	
H. siccans Drechs.	+	unchanged	+	+
H. sorghicola LeFebvre &	—		+	
H. teres Sacc.	—		+	
H. tetramera McK.	+	unchanged	+	+
H. triseptatum Drechs.	+	changed	+	+
H. tritici-repentis Died.	+	changed	—	—
H. tritici vulgaris Nisikado Sherwin	—		—	
H. victoreae Meehan & Murphy	+	unchanged	+	—

[a] Growth type of the recovered cultures remained unchanged.

[b] + indicates recovery; — indicates non-recovery (4 attempts).

[c] Sporulation before and after submergence under sterile mineral oil for 7 years. + indicates typical sporulation; — indicates no sporulation observed.

[d] This isolate was received as *H. ocellum* Faris.

[1] Paper of the Journal Series, New Jersey Agricultural Experiment Station, Rutgers University, the State University of New Jersey, Department of Plant Pathology, New Brunswick.

[2] Respectively, Assistant Research Specialist and Research Specialist, Department of Plant Pathology.

[3] Sherf, A. 1943. A method for maintaining Phytomonas sepedonica in culture for long periods without transfers. Phytopathology 33: 330-332.

[4] Wernham, C. C. 1946. Mineral oil as a fungus culture preservative. Mycologia 38: 691-692.

[5] Buell, C. B., and W. H. Weston. 1947. Application of the mineral oil conservation method to maintaining collections of fungus cultures. Am. J. Botany 34: 555-561.

[6] Wernham, C. C., and H. J. Miller. 1948. Longevity of fungus cultures under mineral oil. Phytopathology 38: 932-934.

[7] Henderson, F. Y. 1954. Report of the director of forest products research for the year 1954. Rep. Forest Prods. Research Board Lond. 1954: 6-49. (Abstr. Rev. Appl. Mycol. 35: 565).

[8] Marathon Corporation, Menasha, Wisconsin.

[9] Henry, B. S. 1947. The viability of yeast cultures preserved under mineral oil. J. Bacteriol. 54: 264.

wine yeasts under mineral oil for 7 years. The recovery of *Helminthosporium* subcultures indicates that other fungi also may be preserved similarly for at least 7 years.—Department of Plant Pathology, Rutgers University, the State University of New Jersey, New Brunswick, New Jersey.

A "Tissue Transplant" Technique for Obtaining Abundant Sporulation of Races of Puccinia graminis var. tritici on Resistant Varieties.[1] E. L. SHARP AND R. G. EMGE.[2] Identification of races of *Puccinia graminis* Pers. var. *tritici* (Eriks. & E. Henn.) Guyot may be hampered by an inability to identify a race that develops as a fleck on any one variety. Since no sporulation occurs, it is not possible to identify such a race by the regular method of single-pustule isolation followed by spore increase and inoculation of differential varieties.

A method has been developed for transplanting such flecks into healthy susceptible leaves to obtain sporulation. The method consists essentially of placing the transplanted tissue containing the desired fleck under the lower epidermis of a susceptible primary leaf.

With a sharp razor blade, a cut was made through the upper epidermis and mesophyll of the healthy leaf, and the lower epidermis was then peeled back from the other tissue (Fig. 1A). The lower epidermis

[1] The authors express appreciation to Dr. C. H. Kingsolver for helpful suggestions given throughout this work.

[2] Chemical Corps, Fort Detrick, Frederick, Maryland. Present addresses, respectively: Montana State College, Bozeman, Mont., and Robt. A. Taft Sanitary Eng. Center, Cincinnati, Ohio.

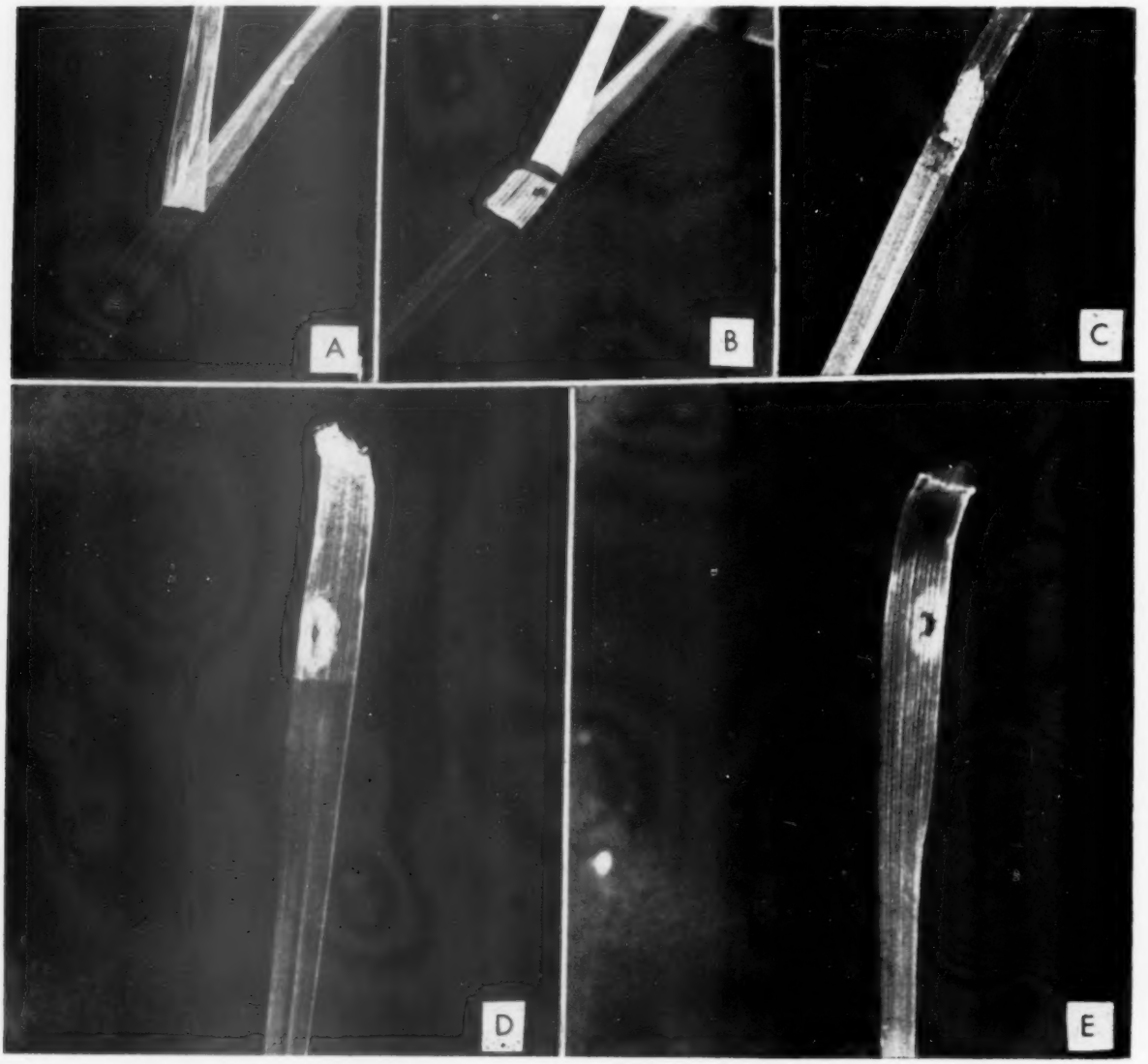

FIG. 1. Illustration of "tissue transplant" technique. A) Lower epidermis separated from other leaf tissue. B) Fleck tissue placed on exposed mesophyll. C) Lower epidermis of healthy leaf replaced in its original position. D) Abaxial leaf surface 7 days after transplanting, showing mycelium growing from the transplant. E) Adaxial leaf surface 7 days after transplanting, showing pustule and surrounding chlorotic area. (U. S. Army photographs.)

was then removed from the desired transplant, which was placed on the healthy leaf so that the exposed mesophylls were placed in contact with each other (Fig. 1B). The lower epidermis of the susceptible leaf was finally placed in its original position, thus covering the transplanted tissue (Fig. 1C).

After 2 days, mycelium of the rust fungus growing from the transplanted tissue and into the susceptible leaf could be observed microscopically. After 5 days, a mass of mycelium was plainly visible surrounding the transplant. A fleck was visible directly above this mycelium on the upper leaf surface. About 7 days after transplanting, a large pustule formed on the upper leaf surface (Fig. 1E). The area of chlorosis on the upper leaf surface corresponded closely to the mycelial mat visible through the lower epidermis (Fig. 1D).

In preliminary testing of the method, race 56 was transplanted as resistant flecks to the susceptible Baart from resistant varieties Lee, Arnautka, Mindum, and Spelmar. In all cases susceptible-type pustules developed in Baart. Immature pustules of race 56 transplanted from Baart to the above resistant varieties gave only the characteristic resistant-type reactions. Flecks produced by race 56 on Lee were successfully transplanted to Baart up to 14 days after inoculation of Lee. Similar tests were made with race 17.

In other tests, Reliance wheat, which produces an immune reaction to race 17, was heavily inoculated with this race. Transplants were made from Reliance to Baart to determine if any living mycelium persisted in the mesophyll of Reliance. Transplants with mesophyll sections gave negative results but susceptible-type pustules were formed when epidermal strips only were transplanted from Reliance to Baart. Microscopic observations confirmed the presence of substomatal vesicles and short infection hyphae on the epidermal strips.

In addition to race identification, the technique may also be used for determining life duration and the extent of mycelial development for various race-variety combinations. With practice, almost all transplants can be established; the technique is simple and no moist chamber is required. Use of this technique with other plant disease organisms is indicated. —Fort Detrick, Frederick, Maryland.

Protection Tests with Clones of Red Clover as an Aid in Identifying Isolates of Bean Yellow Mosaic Virus.[1] STEPHEN DIACHUN AND LAWRENCE HENSON. Red clover plants, *Trifolium pratense* L., with symptoms of virus infection are common in Kentucky as well as in some other clover-growing areas of the United States.[2] Bean yellow mosaic virus is a common cause of disease, but it is often difficult and sometimes impossible to identify the virus on the basis of symptoms in infected field plants. A given strain of the bean yellow mosaic virus may elicit different symptoms in different plants,[3] and different isolates of the virus may cause different symptoms in plants of the same clone (Table 1).

TABLE 1.—*Reaction of 6 selected clones of Kenland red clover to 4 isolates of bean yellow mosaic virus from red clover*

Clone	Symptom induced by isolate			
	204-1	263	266	427
KyC36	Mottle	Mottle	Mottle	Mottle
KyC8-11	Mottle	Mottle	None	None
KyC101	None	None	Mottle	Mottle
KyC71-8	Necrosis	Mottle	Mottle	Mottle
KyC6	Necrosis	Necrosis	Necrosis	Necrosis
KyC5	Necrosis	Mottle	Necrosis	Mottle

This paper reports the use of necrotic-spotting clones of red clover in protection tests to help identify some isolates of bean yellow mosaic virus. An abstract has been published.[4] Corbett recently reported using *Crotalaria spectabilis* Roth. in protection tests to identify strains or isolates of this virus.[5]

The possibility of using a necrotic-spotting clone of red clover for protection tests became apparent when clone KyC71-8, which is necrotic-spotting with the original virus isolate (204-1) used in these studies, was found to become mottled after inoculation with other isolates (Table 1). Accordingly, the following test was conducted. Three plants of clone KyC71-8 were inoculated with virus isolate 263, 3 with isolate 266, and 3 with isolate 427, all originally from naturally infected plants of red clover. Three weeks later, after the viruses were established in the inoculated plants and leaves were mottled, 3 mottled leaves of each plant were rubbed with the challenge virus, 204-1; 3 leaves of each of 3 healthy plants were inoculated at the same time with the same inoculum. Eight days later there were 151 necrotic spots on the 3 healthy plants (respectively, 71, 42, and 38), 1 necrotic spot on one of the plants mottled with virus 427, and no spots on any of the other plants. All 3 of the plants inoculated only with virus 204-1 became systemically necrotic; none of the others did. This test was repeated 4 times, with similar results. Another clone, KyC71, was also tested; the results were similar. These tests show that plants of clones KyC71-8 or KyC71 infected with any one of 3 isolates of bean yellow mosaic virus are protected against both local

[1] The investigation reported is in connection with a project of the Kentucky Agricultural Experiment Station and is published with approval of the Director.

[2] Diachun, S., and L. Henson. 1958. Red clover clones as local-lesion hosts for bean yellow mosaic virus. Phytopathology 48: 369-371.

[3] Diachun, S., and L. Henson. 1956. Symptom reaction of individual red clover plants to yellow bean mosaic virus. Phytopathology 46: 150-152.

[4] Diachun, S., and L. Henson. 1957. A clone of Kenland red clover for cross-protection tests with isolates of yellow bean mosaic virus. (Abstr.) Phytopathology 47: 7.

[5] Corbett, M. K. 1957. Local lesions and cross-protection studies with bean yellow mosaic virus. Phytopathology 47: 573-574.

and systemic infection by isolate 204-1. These tests also give evidence of relationship among the 4 isolates, which for a time were considered different viruses by the authors. Other evidence, such as formation of intranuclear inclusion bodies in cells of infected plants of *Vicia faba* L., and similarity in host range and symptoms produced (except in clones of red clover) supports the revised view.—Kentucky Agricultural Experiment Station, University of Kentucky, Lexington, Kentucky.

BOOK LISTINGS

POTTS, SAMUEL F. 1958. **Concentrated spray equipment, mixtures and application methods.** Dorland Books, Caldwell, New Jersey. 598 p. Price $12.50.

"This text," in the words of the author, "is designed primarily to supply potential users, manufacturers, and distributors [interested in concentrated spraying] with general and concise information on the principles involved in the construction and use of ground and aerial equipment, to discuss various types of equipment for various uses, to discuss the kinds of chemicals and formulations, and to point out the fields of usefulness, hazards, and limitations of each." After a short discussion of terminology, the author presents his subject in considerable detail under the following headings: fundamentals of application, ground spray equipment, aerial equipment, mixtures, control of weeds and woody plants, aerosols, and an appendix covering additional pertinent material. Four pages are devoted to a discussion of the suitability and use of organic and inorganic fungicides in concentrated sprays. No information is presented on the comparative efficacy of concentrated and dilute sprays for the control of plant diseases. The book, however, should prove to be a valuable reference to all agriculturists interested in aerial or ground applications of concentrated sprays.

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INTRODUCTION

S. E. A. McCallan[1]

The second Directory,[2] which appears 5 years after the first one, commemorates the 50th anniversary of the founding of The American Phytopathological Society, at the Baltimore meeting of the American Association for the Advancement of Science on December 30, 1908. In keeping with this golden anniversary celebration, the latter part of this introduction presents data and discussion on some phases in the growth of the Society during the past 50 years.

The Directory has been organized in a manner similar to the first one, together with some added information. Charter Members, first published in the 1953 Directory with original addresses, are again listed in commemoration of the 50th Anniversary. "In Memoriam" includes all members of the Society whose decease has been reported since 1953. The year of birth is also included.

The annual meeting is the high light among Society activities. Accordingly, the 50 annual meetings have been tabulated, with dates, places, and other organizations meeting jointly; also, the number of abstracts of papers submitted for presentation, and the total membership of the Society (usually recorded at the end of the year). For the first thirty-three years, the Society met consistently with the American Association for the Advancement of Science. Since World War II, it has also met alone; with other societies, such as the Potato Association of America; and, more frequently, with the American Institute of Biological Sciences. Cincinnati leads the convention cities, with 5 meetings, and Columbus is next, with 4 meetings, while 3 meetings each have been held in Boston, New York, Philadelphia, and Pittsburgh.

The list of past officers, editors-in-chief, and councilors-at-large has been brought up to date. Until 1941, in accord with the annual meetings, they held office on essentially a calendar basis, and this was indicated by the designation of a single year. This designation has been continued for simplicity, even though meetings are now often held at other times and the term of office will usually have begun in December or earlier in the year before the one indicated. The precise term of office may be ascertained from the list of annual meetings.

A new feature is a complete list of all Divisional Officers, including Councilors, together with time and place of the annual divisional meetings. The geographical areas of the Divisions are also given.

The Society organization for 1958 is detailed since, though the names will change, the general type of organization, committees, and so on that portray the activities of the Society are believed to be of lasting interest. Our industrial Sustaining Associates, who contribute generously to the support of the Society, are listed.

The main body of the Directory is the "Members of The American Phytopathological Society," which gives an alphabetical listing of names, with titles, addresses, and fields of interest. This information was obtained in response to a request sent in December, 1957, to the active list of 1901 members, and later to 117 new members. Post cards were returned by 1682. For those members who did not reply, only the mailing address for PHYTOPATHOLOGY is available. A few late returns, received after March 25, are given in a supplementary list, closing the section. The words "Charter," "Life," "Patron," and "Emeritus" in parentheses after a member's name indicate the form of membership. Insofar as is known, there are believed to be 42 living Charter members. From 1916 to 1936, Life membership was available to those who contributed a total of $100 in 10 consecutive payments; a single payment of the sum constituted a Patron. There are at present 100 Life members, including 2 Patrons. Emeritus memberships were initiated in 1949, essentially for retired members of at least 20 years' active membership, who retain all rights and privileges and are exempt from dues. There are presently 20 Emeritus members. Student members are accredited students, or enlisted personnel in the armed forces, who pay reduced dues but retain all rights and privileges. Student members now total 262.

The Geographical Index of Members lists all members by states and foreign countries. Institutions at which there are 3 or more members are listed separately under the state.

The Constitution of The American Phytopathological Society, revised to January 1, 1958, is given. It may be noted that the original Constitution was published in 1913 in PHYTOPATHOLOGY, and revised editions appeared in 1917, 1925, 1930, 1935, 1940, 1947, and 1953.[3]

GROWTH OF THE SOCIETY IN INSTITUTIONS AND COUNTRIES.—During the Society's life there have appeared, as integral parts or supplements of PHYTOPATHOLOGY, Lists of Members in 1913, 1917, 1925, 1932, 1935, and 1948, and the first Directory in 1953.[4] These total memberships are necessarily fewer than those reported at the end of the year, when membership is at its maximum. From these lists there may be obtained an intimate view of the growth of the Society, and also of the profession of plant pathology in the U. S. Department

[1] Acknowledgment is made to the Boyce Thompson Institute for Plant Research, Inc., Yonkers, New York, for providing time and facilities, and to Dr. Saul Rich, Business Manager of PHYTOPATHOLOGY, for cooperation in furnishing various data on membership.

[2] Published as Section 2 of PHYTOPATHOLOGY, Volume 48, Number 5, May, 1958.

[3] References, respectively: **3**: [2 pp.], 1913; **7**: [2 pp.], 1917; **15**: 618-624, 1925; **20**: 1007-1011, 1930; **25**: 445-449, 1935; **30**: 984-987, 1940; **37**: 265-269, 1947; and **43**: No. 5, Sect. 2, D55-D58, 1953.

[4] Respectively cited: **3**: [3-16]; **7**: [8-26]; **15**: 625-649; **22**: No. 9, Supp. [22 pp.]; **25**: No. 5, Supp. [22 pp.]; **34**: No. 9, Supp. [36 pp.]; **38**: No. 8, Sect. 2 [34 pp.]; and **43**: No. 5, Sect. 2, D1-D58.

of Agriculture, the various universities and colleges, and other institutions. Similar information may be obtained for the different countries represented in our membership. These data, obtained as nearly as possible at 10-year intervals, are presented in Table 2. The institutions and industries of the United States are listed in order of the current number of members. For practicality, institutions with fewer than 5 members are grouped under the headings "Other Colleges, etc." or "Other Industries" or "Miscellaneous." The last category includes retired and unaffiliated members as well as members whose institutional affiliations could not be readily determined even by recourse to standard biographical directories and lists of workers in the agricultural sciences. Sustaining Associates are included in the industrial membership. Members at field or other stations are included under the heading of the main institution. The titles and addresses as given by 5 per cent of the members indicate a cooperative position with both a state college or experiment station and the U. S. Department of Agriculture. These members are listed under both categories. However, it is not to be inferred that such information is complete, since, in general, only the data furnished by the members were used. The 1935 and 1948 Lists of Members did not include information on cooperative positions.

The very large role played in the beginning by members affiliated with the U. S. Department of Agriculture is evident: they constituted 25 per cent of the charter membership, and today furnish 14 per cent. Cornell University, including the Geneva Station, provided 12 per cent of the original membership, and among the state colleges and experiment stations this lead was generally maintained until the fourth decade. Here the lead was taken by the membership from the various branches of the University of California, the most actively growing center in the Society. The universities of Wisconsin and Minnesota consistently furnished large and active membership groups within the Society, and enter the sixth decade in third and tied for fifth as to size. The large increase in membership during the past decade at the University of Florida and the State College of Washington, together with their respective branch stations, and at North Carolina State College is to be noted.

Among the foreign countries providing membership, our neighbor Canada is preeminent, and has consistently furnished some 5 per cent of the membership. The interest of the Canadians in our Society was also demonstrated by the Canadian Division during the second decade, 1918 to 1928. The territory of Hawaii and Commonwealth of Puerto Rico have also maintained an active and consistent interest in the Society. The numbers in parentheses following the entries for "Other Countries" in Table 2, are the number of different countries. India, Mexico, and Brazil have provided large groups of membership among the foreign countries. During the fourth decade the membership from Japan was the second largest; in 1948 there was but 1 member, but it is beginning to build again. At present there are 260 members from 51 different foreign countries, constituting some 14 per cent of total membership.

THE FIRST FIFTY PRESIDENTS.—The election to President is the highest honor that the Society can bestow on a member. In this anniversary edition of the Directory it is appropriate to consider the biographical data on our presidents, and to note trends. There is a highly significant trend toward older presidents. The average age of the first 10 presidents was 45.6 years. This has increased for the presidents of each succeeding decade, to 53.2 for the last decade.

The presidents received their terminal graduate training and most advanced degrees from 23 different educational institutions, but two thirds of the degrees were conferred by 7 institutions: Wisconsin and Cornell, 8 each; Minnesota, 6; University of Michigan, 4; Harvard, 3; and George Washington University and Iowa State College, 2 each.

A somewhat similar concentration may be noted in the 22 different institutions from the staffs of which the 50 presidents were chosen: U. S. Department of Agriculture, 11; Cornell and Wisconsin, 5 each; Minnesota, 4; West Virginia, 3; and 2 each from the University of California, Canada Department of Agriculture, Connecticut Agricultural Experiment Station, Iowa State College, and State College of Washington.

It is perhaps commonly felt that institutions with the larger memberships have a disproportionate share of presidents chosen from their staffs. But, dividing the institutions, including Canada as a unit, into 5 classes based on relative size of membership showed that there is surprisingly little difference in the ratio of presidents to membership for the four larger groups (Table 1), though somewhat more presidents have been selected from large or medium-size groups than from the very large U. S. Department of Agriculture or the small groups. However, no presidents have been chosen from the very small units. While this group does include the institutions with the very small number of members, it also includes unaffiliated members, foreign members exclusive of Canada, and many individuals for whom plant pathology may not be a primary interest. It can be concluded that, above a very small minimum-size membership, the presidents are chosen from institutions on a basis approximately proportional to membership.

Before 1940 there appears to have been considerable effective interest among the members in proposing candidates for president, since almost half were elected without having served as vice-president. However, beginning in 1940, 95 per cent of the presidents have been elected to office directly from the vice-presidency. If this sequence has become more or less the custom, it might be well to recognize it as such, modifying the formal election procedures accordingly.

TABLE 1.—*Selection of presidents from institutions based on relative size of membership*

Size of institution by % of membership	Number of institutions	Total % of membership	Number of presidents	Ratio of presidents to total % of membership
Very large: >10	1	19.7	11	0.58
Large: 3–10	5	21.4	18	0.84
Medium: 1–3	11	14.2	12	0.84
Small: 0.3–1	26	15.4	9	0.58
Very small: <0.3	remainder	29.3	0	0

INCREASE IN MEMBERSHIP AND NUMBERS OF PAPERS PRESENTED AT ANNUAL MEETINGS.—The growth curve of membership was shown graphically in the first Directory. With the exception of the depression years, the growth rate was fairly constant from 1909 to 1940, averaging a net increase of about 30 members per year. From a low point in 1943, during World War II, a quick recovery had been made by 1947, and membership increased by 147 members the following year. It now appears that the net growth rate since then is about 45 members per year, or 50 per cent more than that for the first 3 decades.

The presentation of original research papers at the annual meetings is a good index of the vigor of a scientific society. The number of papers presented yearly, as measured by abstracts accepted, is shown in Fig. 1. While there is considerable yearly fluctuation at some periods, due to such factors as location and time of meeting, drops were marked during World Wars I and II, and, to a lesser extent, during the depression. The over-all trend shows an increase of about 2 papers per year from 1909 to 1940. Again from a low point in 1943, a marked increase in the number of papers is also evident. This reached the high point—250 papers—at the Cincinnati meeting of 1956. The current increase is at an average rate of about 8 papers per year, the equivalent of at least an additional paper session every second meeting.

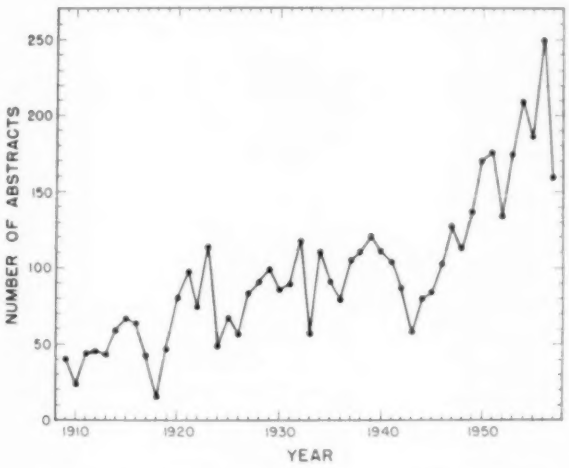

FIG. 1. Volume of papers presented at annual meetings.

The comparison of rate of growth of membership and of papers presented may be made on the basis of papers per 100 members. The average for each of the five decades is 17, 13, 11, 8, and 10. Thus the high rate of paper presentation by the members of a young Society has gradually fallen off, as might be expected. The low during World War I, of about 7, is masked by the preceding high production for this first decade. The fourth decade, 1939-48, is low because of World War II. This, of course, is a reduction in paper presentation below that due to fall in membership. However, since then there has been a recovery, for the current rate is now about equivalent to that of the third decade.

The continuing growth of the Society and presentation of papers, both at an undiminishing rate, undoubtedly presents problems of program and space arrangement at the annual meetings, as was emphasized in the poll of opinion conducted by President George W. Fischer, in 1957.[5] We are fortunate to have this problem, for it is evidence of healthy growth.

[5] Phytopathology 48: 111. 1958.

TABLE 2.—*Growth of membership in the Society, in institutions and countries*

Institution or country	1909	1917	1925	1935	1948	1958
U. S. Dept. Agr. Washington, D. C.	33	79	81	82	15	9
U. S. Dept. Agr. Beltsville, Md.				14	60	57
U. S. Dept. Agr. Other stations		13	22	48	89	86
U. S. Dept. Agr. Cooperative		12	37	—	—	99
U. S. Dept. Agr. Total	33	104	140	144	50	53
University of California	2	8	20	43	164	251
University of Wisconsin	4	16	31	25	65	120
Cornell University	16	32	31	46	54	81
University of Florida	3	5	8	16	50	64
University of Minnesota	2	7	22	30	23	53
State College of Washington	1	5	12	9	21	40
North Carolina State College	2	5	2	3	14	33
Michigan State University	2	4	11	6	17	30
Ohio State University	2	6	15	8	21	29
Louisiana State University	1	3	2	5	25	28
Oregon State College	1	3	7	11	15	27
Pennsylvania State University	1	3	12	6	14	26
Purdue University	2	9	9	11	15	23
Iowa State University	1	3	11	14	17	22
Texas Agr. & Mech. College	1	2	1	3	11	22
University of Illinois	1	5	10	7	14	19
Clemson Agricultural College	2	2	4	2	10	16
Colorado State University		1	3	2	6	16
University of Nebraska	4	2	8	7	10	16
University of Georgia	1	2	2	4	13	15
University of Arkansas		2	3	4	5	14
University of Maryland	1	3	6	6	11	14
Rutgers University	1	7	4	6	7	14
Virginia Polytechnic Institute	1	2	8	7	9	14
University of Idaho		1	4	4	6	13
Oklahoma University		1	2	2	6	13
Boyce Thompson Institute			4	5	8	12
California Dept. Agriculture				3	8	12
E. I. du Pont de Nemours & Co.			1	4	9	12
Kansas State College		4	6	7	10	12
Connecticut Agr. Expt. Station	1	2	4	4	7	11
Mississippi State College	1		3	2	4	11
University of Missouri	1	5	2	3	11	11
Shell Chemical Co.					3	11
University of Tennessee	1	2	6	4	7	11
University of Wyoming				2	3	11
Alabama Polytechnic Institute		2	4		7	9
Dow Chemical Co.					2	9
Fort Detrick					2	9
South Dakota State College			1	1	2	9
University of West Virginia	2	3	6	8	12	9
American Cyanamid Co.					8	8
University of Arizona			1	4	7	8
University of Kentucky	2		2	3	8	8
University of Massachusetts		5	4	10	9	8
Rohm & Haas Co.					3	8
California Packing Corp.				1	5	7
California Spray-Chemical Corp.					3	7
Illinois Natural History Survey			1		4	7
Olin Mathieson Chemical Corp.						7
Campbell Soup Co.					3	6
University of Delaware	2	2	3	1	6	6
University of Maine	2	4	3	6	6	6
Stauffer Chemical Co.						6
Food Machinery Corp.			1	1	4	5
Merck & Co.					2	5
New Mexico Coll. Agr. & Mech. Arts.				1	3	5
Utah State University		2	1	2	4	5
Other colleges, research inst., etc.	20	47	75	93	85	84
Other industries		9	15	15	84	129
Miscellaneous	7	34	42	41	162	146
Total United States	124	352	536	652	1174	1597
Hawaii		1	5	6	18	14
Puerto Rico		3	8	4	4	7
Canada	5	15	43	40	47	83
Other countries	1	14 (10)	46 (25)	89 (25)	137 (38)	179 (52)
Grand total	130	385	638	791	1380	1880

ANNUAL MEETINGS, NUMBER OF ABSTRACTS OF PAPERS PRESENTED, AND TOTAL MEMBERSHIP

NO.	DATE	PLACE	JOINT WITH	ABSTRACTS	MEMBERSHIP[g]
1	1909 Dec. 30–31	Boston, Mass.	AAAS[a]	40	130
2	1910 Dec. 28–30	Minneapolis, Minn.	AAAS	23	167
3	1911 Dec. 27–28	Washington, D. C.	AAAS	43	249
4	1912 Dec. 31–Jan. 3	Cleveland, Ohio	AAAS	45	240
5	1913 Dec. 30–Jan. 2	Atlanta, Ga.	AAAS	42	271
6	1914 Dec. 29–Jan. 1	Philadelphia, Pa.	AAAS	58	293
7	1915 Dec. 28–31	Columbus, Ohio	AAAS	66	335
8	1916 Dec. 27–30	New York, N. Y.	AAAS	63	367
9	1917 Dec. 28–Jan. 1	Pittsburgh, Pa.	AAAS	42	387
10	1918 Dec. 23–28	Baltimore, Md.	AAAS	15	400
11	1919 Dec. 29–Jan. 2	St. Louis, Mo.	AAAS	47	452
12	1920 Dec. 27–31	Chicago, Ill.	AAAS	80	495
13	1921 Dec. 27–30	Toronto, Ont.	AAAS	97	539
14	1922 Dec. 27–30	Boston, Mass.	AAAS	74	553
15	1923 Dec. 27–Jan. 1	Cincinnati, Ohio	AAAS	113	557
16	1924 Dec. 29–Jan. 1	Washington, D. C.	AAAS	48	606
17	1925 Dec. 28–31	Kansas City, Mo.	AAAS	67	603
18	1926 Dec. 28–31	Philadelphia, Pa.	AAAS	56	650
19	1927 Dec. 28–30	Nashville, Tenn.	AAAS	83	685
20	1928 Dec. 28–31	New York, N. Y.	AAAS	91	794
21	1929 Dec. 28–31	Des Moines, Iowa	AAAS	98	769
22	1930 Dec. 30–Jan. 1	Cleveland, Ohio	AAAS	86	828
23	1931 Dec. 29–31	New Orleans, La.	AAAS	89	836
24	1932 Dec. 28–30	Atlantic City, N. J.	AAAS	117	827
25	1933 Dec. 28–30	Boston, Mass.	AAAS	57	774
26	1934 Dec. 27–29	Pittsburgh, Pa.	AAAS	110	798
27	1935 Dec. 31–Jan. 3	St. Louis, Mo.	AAAS	91	832
28	1936 Dec. 28–31	Atlantic City, N. J.	AAAS	79	965
29	1937 Dec. 27–30	Indianapolis, Ind.	AAAS	105	1053
30	1938 Dec. 27–30	Richmond, Va.	AAAS	111	1077
31	1939 Dec. 27–30	Columbus, Ohio	AAAS	121	1082
32	1940 Dec. 27–31	Philadelphia, Pa.	AAAS	111	1128
33	1941 Dec. 29–Jan. 1	Dallas, Tex.	AAAS	103	1118
34	1943 Feb. 12–14	Columbus, Ohio	[b]	87[f]	1085[h]
35	1943 Dec. 4–6	Columbus, Ohio	ESA[c]	58	1060[i]
36	1944 Dec. 9–11	Cincinnati, Ohio	Alone	80	1089[j]
37	1946 Mar. 27–30	St. Louis, Mo.	AAAS	84	1100
38	1946 Dec. 28–30	Cincinnati, Ohio	AAAS	102	1304
39	1947 Dec. 28–30	Chicago, Ill.	AAAS	127	1350
40	1948 Dec. 6–8	Pittsburgh, Pa.	PAA[d]	112	1497
41	1949 Dec. 28–30	New York, N. Y.	AAAS	136	1575
42	1950 Dec. 1–3	Memphis, Tenn.	PAA	169	1533
43	1951 Dec 9–12	Cincinnati, Ohio	PAA, ESA	176	1623
44	1952 Sept. 7–10	Ithaca, N. Y.	AIBS[e]	133	1633
45	1953 Sept. 6–10	Madison, Wis.	AIBS	174	1736
46	1954 Aug. 24–27	Estes Park, Colo.	PAA	208	1737
47	1955 Dec. 27–30	Atlanta, Ga.	AAAS	185	1735
48	1956 Dec. 5–8	Cincinnati, Ohio	PAA	250	1846
49	1957 Aug. 25–29	Palo Alto, Calif.	AIBS	158	1901
50	1958 Aug. 24–28	Bloomington, Ind.	AIBS		

[a] American Association for the Advancement of Science.
[b] Council and War Committee only.
[c] Entomological Society of America and American Associa tion of Economic Entomologists.
[d] Potato Association of America.
[e] American Institute of Biological Sciences.
[f] Abstracts were accepted, and later published for AAAS meeting in New York, scheduled for Dec. 27–31, 1942, and later canceled.
[g] Membership is on or about Dec. 31, unless otherwise indicated.
[h] February 12, 1943.
[i] December 6th.
[j] December 11th.

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THE DIVISIONS

The Officers and Councilors of the various Divisions are tabulated below, together with the time and place of the annual meetings at which they were elected.[6] The Canadian Division was active from 1918 until 1929, when it was automatically dissolved on the formation of the Canadian Phytopathological Society. In accord with the Constitution, each Division is represented on the Society Council by one Councilor. The Canadian Division, however, by their rules, sent their President as representative on the Council. These representatives were recorded in Phytopathology as the Canadian Councilors, and subsequently so in the first Directory. In the beginning, a similar situation seems to have held in the Pacific and Southern Divisions since formal election of Councilors does not appear to have taken place until 1922 and 1920, respectively. The immediate Past President of the Canadian Division also served as ex-officio member of the five-man Canadian Council.

[6] Acknowledgment for cooperation in seeking out the information in sometimes obscure records is made to divisional Secretaries, present and past: Pacific, W. A. Kreutzer and G. A. Zentmyer; Southern, W. J. Martin, G. B. Lucas, J. A. Lyle, and Past-president G. F. Weber; Northeastern, Thomas Sproston, Jr., and Frank L. Howard; Potomac, J. C. Moseman; North Central, E. W. Hanson; and Canadian, D. S. MacLaclan, Secretary-Treasurer of the Canadian Phytopathological Society.

At least 2 of the Divisions are known to have been formed from earlier associations of plant pathologists. The Pacific Division was formed from the "Western American Phytopathological Society," created a year earlier, in 1914. From a group of "New England Plant Pathologists" that had met in 1931, 1937, and 1940, the New England Division was created in 1941, and enlarged to the present Northeastern Division in 1946.

The geographical areas embraced by the various Divisions, which, as may be seen, can overlap, are as follows:

Pacific—California, Oregon, Washington, Arizona, Utah, Nevada, Idaho, Montana, British Columbia, Alaska, and Hawaii; and, informally, New Mexico, Colorado, and Wyoming.

Southern—Virginia, North Carolina, South Carolina, Georgia, Florida, Alabama, Mississippi, Tennessee, Kentucky, Arkansas, Louisiana, Texas, Oklahoma, New Mexico, and Arizona.

Northeastern—Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, and Pennsylvania.

Potomac—District of Columbia, Maryland, Delaware, northern Virginia, and West Virginia.

North Central—Ohio, Indiana, Michigan, Wisconsin, Illinois, Kentucky, Missouri, Iowa, Minnesota, North Dakota, South Dakota, Nebraska, and Kansas.

POTOMAC DIVISION

Annual Meeting	*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1944 Feb. 23–24*	W. J. Zaumeyer	H. B. Humphrey	T. F. Manns	H. P. Barss
1945 Feb. 20–21	T. F. Manns	E. E. Clayton	V. F. Tapke	R. J. Haskell
1946 Feb. 19–20	E. E. Clayton	V. F. Tapke	W. F. Jeffers	R. J. Haskell
1947 Feb. 19–20	V. F. Tapke	W. F. Jeffers	W. W. Diehl	E. E. Clayton
1948 Feb. 11–12	W. F. Jeffers	C. L. Lefebvre	J. B. Demaree	P. R. Miller
1949 Feb. 23–24	C. L. Lefebvre	C. E. Cox	J. B. Demaree	P. R. Miller
1950 March 9–10	C. E. Cox	S. P. Doolittle	J. B. Demaree	C. L. Lefebvre
1951 Feb. 27–28	S. P. Doolittle	J. B. Demaree	H. R. Thomas	C. E. Cox
1952 Feb. 26–27	J. B. Demaree	Curtis May	H. R. Thomas	C. E. Cox
1953 Feb. 19–20	Curtis May	J. W. Heuberger	H. R. Thomas	W. J. Zaumeyer
1954 March 4–5	J. W. Heuberger	H. R. Thomas	L. O. Weaver	W. J. Zaumeyer
1955 March 3–4	H. R. Thomas	J. G. Leach	L. O. Weaver	W. D. McClellan
1956 March 1–2	J. G. Leach	K. W. Kreitlow	L. O. Weaver	W. D. McClellan
1957 Feb. 28–Mar. 1	K. W. Kreitlow	L. O. Weaver	J. G. Moseman	J. W. Heuberger

* All meetings held at Beltsville, Md., except for second day, 1957, at Fort Detrick, Md.

NORTH CENTRAL DIVISION

Annual Meeting		*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1948 July 22–23	St. Paul, Minn.	H. C. Young, Sr.	J. C. Walker	M. F. Kernkamp	W. F. Buchholtz
1949 June 27–28	Lincoln, Nebr.	R. M. Caldwell	L. E. Melchers	M. F. Kernkamp	W. F. Buchholtz
1950 June 22–24	East Lansing, Mich.	C. S. Reddy	H. H. Thornberry	M. F. Kernkamp	W. Q. Loegering
1951 June 18–20	Madison, Wis.	H. H. Thornberry	M. F. Kernkamp	J. R. Shay	E. W. Hanson
1952 Aug. 11–12	Lafayette, Ind.	M. F. Kernkamp	J. R. Vaughan	J. R. Shay	E. W. Hanson
1953 June 15–16	Urbana, Ill.	Benj. Koehler	Donald Cation	J. R. Shay	E. W. Hanson
1954 June 28–29	Ames, Iowa	Donald Cation	C. O. Johnston	M. B. Linn	J. R. Shay
1955 Aug. 8–10	Wooster, Ohio	C. O. Johnston	J. J. Christensen	M. B. Linn	J. R. Shay
1956 June 5–6	Manhattan, Kans.	J. J. Christensen	W. B. Allington	M. B. Linn	W. H. Sill, Jr.
1957 July 12–13	St. Paul, Minn.	G. H. Boewe	W. B. Allington	E. W. Hanson	W. H. Sill, Jr.

PACIFIC DIVISION

Annual Meeting		*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1914[a] June 2	Davis, Calif.	R. E. Smith	H. S. Jackson	W. T. Horne	
1915[b] Aug. 3–5	Berkeley, Calif.	H. S. Jackson	J. T. Barrett	W. T. Horne	
1916 Dec. 28–29	Berkeley, Calif.	H. P. Barss	James McMurphy	W. T. Horne	
1917 No meeting		"	"	"	
1918 March 30	Berkeley, Calif.	F. D. Heald	E. P. Meinecke	W. T. Horne	
1919 April 25–26	Portland, Ore.	"	"	"	F. D. Heald
1920 June 17–18	Seattle, Wash.	H. S. Reed	J. W. Hotson	S. M. Zeller	H. S. Reed
1921 Aug. 4–6	Berkeley, Calif.	"	"	"	"
1922 June 22–24	Salt Lake City, Utah	S. M. Zeller	J. P. Bennett	Chas. Hungerford	F. D. Heald
1923 Sept. 18–21	Los Angeles, Calif.	"	"	"	"
1924 Aug. 26–29	Penticton, B. C.	J. W. Eastham		C. E. Owens	S. M. Zeller
1925 June 22–24	Corvallis, Ore.	"		"	"
1926 June 16–19	Oakland, Calif.	W. S. Ballard	D. G. Milbrath	B. A. Rudolph	W. T. Horne
1927 June 23	Reno, Nev.	"	"	"	"
1928 June 13–16	Claremont, Calif.	J. W. Hotson	Eubanks Carsner	B. A. Rudolph	J. T. Barrett
1929 June 19–22	Berkeley, Calif.	"	"	"	"
1930 June 19–20	Eugene, Ore.	Eubanks Carsner	J. M. Raeder	B. A. Rudolph	C. E. Owens
1931 June 16	Pasadena, Calif.	"	"	"	"
1932 June 16–17	Pullman, Wash.	H. E. Morris	F. P. McWhorter	B. A. Rudolph	J. M. Raeder
1933 June 14–15	Salt Lake City, Utah	"	"	B. A. Rudolph	"
1934 June 18–19	Berkeley, Calif.	C. O. Smith	C. W. Bennett	B. A. Rudolph	T. E. Rawlins
1935 June 26–27	Los Angeles, Calif.	C. E. Owens	"	"	"
1936 June 16–20	Seattle, Wash.	B. A. Rudolph	C. W. Bennett	L. D. Leach	J. W. Hotson
1937 June 23–26	Denver, Colo.	C. W. Bennett	B. F. Dana	"	"
1938 June 21–23	San Diego, Calif.	W. T. Horne	"	L. D. Leach	Eubanks Carsner
1939 June 27–30	Palo Alto, Calif.	B. F. Dana	T. E. Rawlins	"	"
1940 June 19–22	Seattle, Wash.	T. E. Rawlins	R. B. Streets	C. E. Yarwood	N. J. Giddings
1941 June 18–21	Pasadena, Calif.	R. B. Streets	L. D. Leach	"	"
1942 June 17–19	Salt Lake City, Utah	L. D. Leach	B. L. Richards	C. E. Yarwood	G. A. Huber
1943 No meeting		"	"	"	E. E. Wilson
1944[d] June 26–28	Corvallis, Ore.	B. L. Richards	H. R. McLarty	G. W. Fischer	L. D. Leach
1945 No meeting		"	"	"	"
1946[d] June 24–27	Berkeley, Calif.	C. E. Yarwood	L. D. Cochran	G. W. Fischer	M. W. Gardner
1947[d] June 17–19	San Diego, Calif.	L. C. Cochran	G. W. Fischer	G. A. Zentmyer	"
1948[d] June 22–25	Berkeley, Calif.	G. W. Fischer	W. C. Snyder	G. A. Zentmyer	L. C. Cochran
1949[d] June 16–18	Vancouver, B. C.	W. C. Snyder	E. C. Blodgett	"	"
1950[d] June 21–23	Salt Lake City, Utah	E. C. Blodgett	W. B. Hewitt	G. A. Zentmyer	S. M. Dietz
1951 June 19–21	Los Angeles, Calif.	W. B. Hewitt	E. K. Vaughan	"	"
1952 June 19–21	Corvallis, Ore.	E. K. Vaughan	G. L. Stout	R. A. Young	W. B. Hewitt
1953 June 17–19	Santa Barbara, Calif.	G. L. Stout	G. A. Zentmyer	R. A. Young	"
1954[e] Aug. 25–27	Estes Park, Colo.	G. A. Zentmyer	S. M. Dietz	R. A. Young	E. C. Blodgett
1955 June 22–24	Pasadena, Calif.	S. M. Dietz	Wm. Newton	R. A. Young	"
1956 June 13–15	Seattle, Wash.	R. A. Young	C. S. Holton	W. A. Kreutzer	Roderick Sprague
1957[e] Aug. 26–28	Palo Alto, Calif.	C. S. Holton	J. B. Kendrick, Sr.	W. A. Kreutzer	J. A. Milbrath

[a] Western American Phytopathological Society; a second meeting was held Dec. 29–30, at Corvallis, Ore.
[b] A second meeting was held Dec. 29, at Riverside, Calif. During the interval, H. S. Jackson left the region, and J. T. Barrett succeeded to the presidency.
[c] "Ditto" marks indicate a 2-year term of office.
[d] Term of office on a calendar basis, beginning the following January.
[e] Met with the parent Society.

CANADIAN DIVISION

Annual Meeting		*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1918 Dec. 6–7	Guelph, Ont.	J. E. Howitt	W. A. McCubbin	R. E. Stone	W. P. Frazer
					P. A. Murphy
1919 Dec. 11–12	Guelph, Ont.	A. H. R. Buller	J. H. Faull	R. E. Stone	F. L. Drayton
1920 Dec. 9–10	Guelph, Ont.	J. H. Faull	W. H. Rankin	R. E. Stone	B. T. Dickson
1921[a] Dec. 27–30	Toronto, Ont.	W. H. Rankin	W. P. Frazer	R. E. Stone	
1922 Dec. 7–8	Ste. Anne de Bellevue, Que.	W. P. Frazer	B. T. Dickson	R. E. Stone	W. T. MacClement[b] G. C. Cunningham
1923 Dec. 20–21	Kingston, Ont.	B. T. Dickson	W. T. MacClement	R. E. Stone	G. H. Berkeley
1924 Dec. 22–23	Ottawa, Ont.	W. T. MacClement	R. E. Stone	D. R. Sands	John Dearness
1925 Dec. 21–22	St. Catherines, Ont.	R. E. Stone	John Dearness	D. R. Sands	D. L. Bailey
1926 Dec. 21–22	Guelph, Ont.	John Dearness	D. L. Bailey	D. R. Sands	W. P. Frazer
1927 Dec. 20–21	Winnipeg, Man.	D. L. Bailey	F. L. Drayton	D. R. Sands	G. R. Bisby
1928 Dec. 20–21	Guelph, Ont.	F. L. Drayton	G. H. Berkeley	D. R. Sands	J. G. Coulson
1929 Dec. 19	Ottawa, Ont.	Division dissolved on formation of Canadian Phytopathological Society			

[a] Met with parent society.
[b] Additional Councilor elected to fill term of retired President, no longer resident in Canada.

NORTHEASTERN DIVISION

Annual Meeting		*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1941 June 25[a]	Windsor, Conn.	J. G. Horsfall		F. L. Howard	F. L. Howard
1942 Feb. 9	Providence, R. I.	B. F. Lutman	E. F. Guba	M. C. Richards	F. L. Howard
1943 Feb. 2	New Haven, Conn.	E. F. Guba	R. Marshall	M. C. Richards	O. C. Boyd
1944 Feb. 24	Waltham, Mass.	M. C. Richards	D. Folsom	G. A. Zentmyer	O. C. Boyd
1945 Feb. 15–16	Waltham, Mass.	D. Folsom	F. L. Howard	T. Sproston, Jr.	M. C. Richards
1945 Dec. 4–5	New Haven, Conn.	F. L. Howard	P. J. Anderson	T. Sproston, Jr.	M. C. Richards
1946 Nov. 26–27[b]	Amherst, Mass.	S. E. A. McCallan	M. T. Hilborn	W. T. Schroeder	T. Sproston, Jr.
1947 Nov. 25–26	Poughkeepsie, N. Y.	O. C. Boyd	D. H. Palmiter	L. M. Black	S. E. A. McCallan
1948 Dec. 6–8[c]	Pittsburgh, Pa.	D. H. Palmiter	E. M. Stoddard	L. M. Black	S. E. A. McCallan
1949 Dec. 28–30[c]	New York, N. Y.	E. G. Rex	E. G. Rex	S. G. Younkin	M. T. Hilborn
1950 Dec. 19–20	W. Springfield, Mass.	E. M. Stoddard	J. M. Hamilton	S. G. Younkin	L. M. Black
1951 Nov. 6–7	W. Springfield, Mass.	J. M. Hamilton	S. G. Younkin	J. S. Tidd	L. M. Black
1952 Nov. 6–7	W. Springfield, Mass.	S. G. Younkin	J. S. Tidd	A. B. Burrell	F. O. Holmes
1953 Nov. 5–6	W. Springfield, Mass.	J. S. Tidd	A. B. Burrell	A. C. Tarjan	F. O. Holmes
1954 Nov. 4–5	W. Springfield, Mass.	A. B. Burrell	A. C. Tarjan	B. H. Davis	E. G. Rex
1955 Nov. 3–4	W. Springfield, Mass.	F. O. Holmes	B. H. Davis	B. A. Friedman	E. G. Rex
1956 Nov. 8–9	W. Springfield, Mass.	B. H. Davis	B. A. Friedman	A. E. Rich	D. S. Welch
1957 Nov. 7–8	W. Springfield, Mass.	B. A. Friedman	A. E. Rich	J. E. Livingston	D. S. Welch

[a] Formation of New England Division from the "New England Plant Pathologists."
[b] First Meeting of Division enlarged to Northeastern.
[c] Met with parent society.

SOUTHERN DIVISION

Annual Meeting		*President*	*Vice-President*	*Sec.-Treas.*	*Councilor*
1918 Jan. 30–Feb. 1	Atlanta, Ga.	H. W. Barre	C. W. Edgerton	H. E. Stevens	
1919 Feb. 26–28	Birmingham, Ala.	C. W. Edgerton		J. A. Elliott	H. W. Barre
1920 Feb. 24–25	Atlanta, Ga.	S. H. Essary	J. A. Elliott	J. A. McClintock	H. R. Rosen
1921 Feb. 15–17	Lexington, Ky.	J. A. Elliott		J. J. Taubenhaus	C. W. Edgerton
1922 Feb. 20–22	Atlanta, Ga.	H. W. Barre	S. H. Essary	J. J. Taubenhaus	L. R. Hesler
1923	No meeting				
1924 Jan. 10–12	Birmingham, Ala.	C. D. Sherbakoff	D. C. Neal	L. E. Miles	L. R. Hesler
1925 Feb. 3–4	Atlanta, Ga.	D. C. Neal		L. E. Miles	C. A. Ludwig
1926 Feb. 3–5	Atlanta, Ga.	S. G. Lehman		W. B. Tisdale	C. A. Ludwig
1927 Feb. 2–4	Atlanta, Ga.	W. B. Tisdale		V. H. Young	C. A. Ludwig
1927 Dec. 28-30[a]	Nashville, Tenn.	B. B. Higgins		V. H. Young	W. B. Tisdale
1928 July 11–13	Experiment, Ga.	R. F. Poole		H. H. Wedgeworth	B. B. Higgins
1929 Feb. 5–7	Houston, Tex.	C. D. Sherbakoff	L. E. Miles		R. F. Poole
1930 Feb. 5–7	Jackson, Miss.	L. E. Miles			R. F. Poole
1931–1933	No meeting			L. E. Miles	L. E. Miles
1934 Jan. 31-Feb. 2	Memphis, Tenn.	D. C. Neal	G. F. Weber	E. C. Tims	V. H. Young
1935 Jan. 31–Feb. 1	Atlanta, Ga.	G. F. Weber	C. W. Edgerton	J. C. McKee	V. H. Young
1936 Feb. 5–7	Jackson, Miss.	C. W. Edgerton	A. N. Brooks	J. C. McKee	G. F. Weber
1937 Feb. 5–7	Nashville, Tenn.	A. N. Brooks	D. C. Neal	J. C. McKee	G. F. Weber
1938 Feb. 2–4	Atlanta, Ga.	G. M. Armstrong	V. H. Young	Luther Shaw	A. N. Brooks
1939 Feb. 1–3	New Orleans, La.	A. G. Plakidas	C. H. Arndt	Luther Shaw	A. N. Brooks
1940 Feb. 7–9	Birmingham, Ala.	C. H. Arndt	A. L. Harrison	Luther Shaw	G. M. Armstrong
1941 Feb. 5, 6, 8	Atlanta, Ga.	A. A. Dunlap	Luther Shaw	I. L. Forbes	G. M. Armstrong
1942 Feb. 4–6	Memphis, Tenn.	S. G. Lehman	W. H. Thorp	I. L. Forbes	A. G. Plakidas
1943–1944	No meeting			I. L. Forbes	A. G. Plakidas
1945–1946	No meeting			I. L. Forbes	C. H. Arndt
1947 Jan. 15–17	Biloxi, Miss.	A. L. Smith	C. J. Nusbaum	W. W. Ray	S. G. Lehman
1948 Feb. 12–14[b]	Washington, D. C.	I. L. Forbes	L. M. Blank	J. A. Lyle	S. G. Lehman
1949 Jan. 31–Feb. 2	Baton Rouge, La.	L. M. Blank	W. A. Jenkins	J. A. Lyle	A. L. Smith
1950 Feb. 9–11	Biloxi, Miss.	W. A. Jenkins	Phares Decker	J. A. Lyle	A. L. Smith
1951 Dec. 1–3[a]	Memphis, Tenn.	Phares Decker	A. L. Harrison	J. A. Lyle	Coyt Wilson
1952 Feb. 4–6	Atlanta, Ga.	J. H. Jensen	A. L. Harrison	J. A. Lyle	Coyt Wilson
1953 Feb. 9–11	New Orleans, La.	S. S. Ivanoff	J. H. Miller	J. A. Lyle	S. J. P. Chilton
1954 Feb. 1–3	Dallas, Texas	J. H. Miller	T. W. Graham	G. B. Lucas	S. J. P. Chilton
1955 Feb. 7–9	Louisville, Ky.	T. W. Graham	J. A. Lyle	G. B. Lucas	J. L. Allison
1956 Dec. 27–30[a]	Atlanta, Ga.	J. A. Lyle	W. W. Hare	G. B. Lucas	J. L. Allison
1957 Feb. 4–6	Birmingham, Ala.	W. W. Hare	G. B. Lucas	W. J. Martin	D. E. Ellis
1958 Feb. 3–5	Little Rock, Ark.	G. B. Lucas	E. M. Cralley	W. J. Martin	D. E. Ellis

[a] Met with parent society.
[b] First day in joint session with Potomac Division, at Beltsville, Md.

The January and February meetings were held in conjunction with the Association of Southern Agricultural Workers. The 1918 and 1921 meetings are believed to have been held with the A.S.A.W., as indicated, but confirmation is not available.

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UNITED STATES

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Self, Raymond Lester
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Cairns, Eldon James
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Lyle, James A.
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Arizona

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California

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MacMillan, Howard Gove
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Milbrath, David Gallus
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Panasenko, Vasil T.
Poole, D. Donald
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Buchenau, George W. Graduate Assistant, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Cereal diseases.)

Buchholtz, Walter F. (Life) Professor of Plant Pathology, Iowa State College, Ames, Iowa. (Root diseases of field crops; virus and other diseases of nursery crops.)

Buckner, David Arnold. Graduate Research Assistant, Botany and Bacteriology Department, Clemson College, Clemson, S. C. (Diseases caused by fungi and viruses.)

Buddenhagen, Ivan W. Plant Pathologist, United Fruit Company, Coto Research Station, Coto, Costa Rica. (Microbial genetics and variability; ecology of fungus and bacterial diseases.)

Bulger, Raymond O. Regional Supervisor, Plant Pest Control Division, Agricultural Research Service, 35 South 5th St., Minneapolis, Minn. (Plant pests, particularly cereal diseases.)

Burchfield, Harry P. Chemist, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Fungicides; nematocides.)

Burke, Douglas W. Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Irrigation Experiment Station, Prosser, Wash. (Vegetable diseases; soil microbiology.)

Burkholder, Walter H. (Life) Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Bacterial diseases of plants.)

Burnett, Harry C. Assistant Plant Pathologist, State Plant Board of Florida, P. O. Box 3777, University Station, Gainesville, Fla. (Diseases of citrus, tropical fruits and orchids.)

Burnette, Barbara Delores. Graduate Research Assistant, Botany Department, Clemson College, Clemson, S. C. (Plant pathology.)

Burrell, Arthur B. (Life) Professor of Plant Pathology, Cornell University Orchard Laboratory, Peru, N. Y. (Fruit diseases, expecially those of nutritional origin; fungicides; spray machinery.)

Burton, Clyde L. 900 No. Harrison Rd., East Lansing, Mich.

Busch, Lloyd V. Professor of Plant Pathology, Department of Botany, Ontario Agricultural College, Guelph, Ontario, Canada. (Vegetable diseases of muck crops.)

Buschner, Carl W. Graduate Teaching Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Diseases of tree fruits; general plant pathology; teaching.)

Butler, Edward E. Assistant Plant Pathologist, Department of Plant Pathology, University of California, Davis, Calif.

Cairns, Eldon James. Nematologist and Professor of Botany, Department of Botany and Plant Pathology, Alabama Polytechnic Institute, Auburn, Ala. (Plant and soil nematodes.)

Calabresa, James V. Plant Pathologist, Electric Steel Elevator Division of Russell-Miller Milling Company, 970 Grain Exchange, Minneapolis 15, Minn. (Cereal diseases.)

Calavan, E. C. Associate Plant Pathologist, Department of Plant Pathology, University of California, Citrus Experiment Station, Riverside, Calif. (Citrus diseases; fungicides; virology.)

Caldis, Panos Demetrius. Director, Agricultural Research, California Packing Corporation, 215 Fremont St., San Francisco, Calif. (Fruit and vegetable diseases; breeding for resistance.)

Caldwell, Ralph M. Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Life Science Building, Purdue University, Lafayette, Ind. (Disease resistance; cereal pathology.)

Callahan, Kemper L. Plant Pathologist, Forest Insect and Disease Laboratory, 335 Prospect St., New Haven, Conn. (Virus diseases of woody plants, particularly those common to forest trees.)

Calpouzos, Lucas. Plant Pathologist, Agricultural Research Service, United State Department of Agriculture, Federal Experiment Station, Maygüez, Puerto Rico. (Diseases of bananas and other tropical crops; action of fungicides.)

Calvert, O. H. Assistant Professor, Department of Field

Crops, 108 Waters Hall, University of Missouri, Columbia, Mo. (Field crop diseases.)

Cameron, H. Ronald. Assistant Professor of Plant Pathology, Assistant Plant Pathologist, Agricultural Experiment Station, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Diseases of orchard crops.)

Campagna, Elzéar. Professor of Plant Pathology, Laboratoire de Botanique, Faculté d'Agriculture, Laval University, Ste-Anne-de-la-Pocatière, Kamouraska, Quebec, Canada. (Potato diseases.)

Campana, Richard John. Associate Plant Pathologist, Illinois Natural History Survey, University of Illinois, Urbana, Ill. (Shade and forest tree diseases.)

Campbell, Leo. Plant Pathologist, Western Washington Experiment Station, Puyallup, Wash. (Diseases of vegetable crops; mint rust; red stele of strawberries.)

Campbell, Robert N. Pathologist, Forest Products Laboratory, Forest Service, United States Department of Agriculture, Madison 5, Wis. (Forest pathology; products pathology; mycology; fungus physiology.)

Campbell, Thomas H. Assistant Professor of Botany, Department of Botany, University of Tennessee, Knoxville, Tenn. (Teaching of plant pathology; mycology.)

Campbell, W. P. Science Service, University of Alberta, Box 913, Edmonton, Alberta, Canada.

Campbell, William A. Research Center Leader, Southern Forest Experiment Station, United States Forest Service, School of Forestry, University of Georgia, Athens, Ga. (Forest pathology; wood-rotting and root disease fungi.)

Cannon, Orson S. Professor and Head of Department, Department of Botany and Plant Pathology, Utah State University, Logan, Utah. (Vegetable diseases and breeding for disease resistance.)

Capoor, S. P. Virus Pathologist, I.A.R.I., Plant Virus Research Laboratory, Agricultural College Estate, Poona 5, Bombay State, India. (Virology.)

Cappellini, Raymond A. Assistant Research Specialist, Department of Plant Pathology, College of Agriculture, New Brunswick, N. J. (Field crop diseases.)

Cardona-Alvarez, Canuto. Director of the Bean Improvement Program, Colombian Ministry of Agriculture, Apartado Nacional 2020, Medellin-Colombia, South America. (Breeding field beans for disease resistance; etiology and epidemiology of dry bean diseases.)

Carlson, Lester W. Graduate Student, Department of Botany and Plant Pathology, Oklahoma State University, Stillwater, Okla. (Vegetable diseases.)

Caron, Omer. Chief of Division of Phytopathology, Department of Agriculture, Quebec, P. Q., Canada. (Phytopathology; plant physiology.)

Carone, Margarita. Havana University, Calzada 307-DPTO 6 Entre H Ei, Vedado, La Habana, Cuba.

Caroselli, N. E. P. O. Box 17, West Kingston, R. I.

Carpenter, J. B. United States Date Garden, P. O. Box 727, Indio, Calif.

Carpenter, Theodore R. Associate Plant Pathologist, State Department of Agriculture, Bureau of Plant Pathology, 900 Mirror Building, 145 South Spring St., Los Angeles 12, Calif. (New plant pest detection and surveys.)

Carter, Edward P. 5505 42nd Ave., Hyattsville, Md.

Carter, James Cedric. Plant Pathologist and Head, Section of Applied Botany and Plant Pathology, State Natural History Survey; Professor of Plant Pathology, University of Illinois, 387 Natural Resources Building, Urbana, Ill. (Tree and shrub diseases; fungicides; antibiotics.)

Carter, R. D. Entomologist, Agricultural Research Department, California Packing Corporation, 850 Thornton St., San Leandro, Calif. (Arthropod-plant disease relationship; agricultural entomology and nematology.)

Carter, Walter. Head of Entomology Department and Senior Scientist, Pineapple Research Institute, Honolulu, Hawaii. (Insects in relation to plant diseases.)

Cartwright, T. E. Assistant Professor of Biophysics, Department of Biophysics, University of Pittsburgh, Pittsburgh 13, Pa. (Virology.)

Carvajal, Fernando. 500 Morningside Rd., Ridgewood, N. J.

Cash, Edith K. Mycologist, United States Plant Industry Station, Beltsville, Md. (Taxonomy of Discomycetes; Alaskan fungi.)

Castillo, Bernardo S. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Cation, Donald. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Fruit diseases; stone fruit virus diseases; fungicides.)

Ceponis, Michael J. Associate Plant Pathologist, United States Department of Agriculture, Market Pathology Laboratory, Room 1022, 641 Washington St., New York 14, N. Y. (Bacterial soft rots; enzymology.)

Cetas, Robert C. Associate Professor of Plant Pathology, Cornell University, Long Island Vegetable Research Farm, Riverhead, Long Island, N. Y. (Vegetable diseases.)

Chakravarti, Bimala P. Graduate Student, Department of Plant Pathology, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Cereal diseases.)

Chamberlain, Donald W. Plant Pathologist, United States Department of Agriculture; Assistant Professor of Plant Pathology, University of Illinois, 243 Davenport Hall, Urbana, Ill. (Soybean diseases; disease resistance; bacterial leafspot diseases.)

Chambers, Ernest Leslie. State Entomologist and Chief, Division of Plant Industry, Wisconsin Department of Agriculture, 448 W. Washington Ave., Madison, Wis. (Plant quarantine and regulatory work.)

Chandler, Webster A. Assistant Plant Pathologist, Department of Plant Pathology, Georgia Experiment Station, Experiment, Ga. (Fruit and vegetable diseases; fungicides.)

Chang, Sang Wook. Dean, College of Agriculture, Kyungpook University, Taegu, Korea. (Field crops.)

Changsri, Winit. Graduate Student of Plant Pathology, University of Florida, Box 3239, University Station, Gainesville, Fla. (Plant pathology and entomology.)

Chao, S. John. Plant Pathologist, Rural Route #2, Clear Lake, Iowa. (Soybean diseases; seed and soil testing.)

Chapman, Richard A. Plant Pathologist, Agronomy Department, Kentucky Agricultural Experiment Station, Lexington, Ky. (Nematode diseases.)

Chen, Der-Fen. Research Assistant, Department of Plant Pathology, University of Minnesota, St. Paul, Minn. (Biochemistry, microorganism.)

Chen, Peter K. Y. Research Assistant and Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Bacterial diseases; virology.)

Cheney, Philip W. Biological Aide, Box 99, Wenatchee, Wash. (Virology.)

Cheo, Pen Ching. Division of Biology, California Institute of Technology, Pasadena, Calif.

Cherewick, W. J. Plant Pathologist, Canada Department of Agriculture Research Laboratory, Box 322, % University of Manitoba, Winnipeg, Manitoba, Canada. (Cereal smuts.)

Chester, K. Starr. Technical Advisor to the President, Alton Box Board Company, Alton, Ill. (Plant disease losses; cereal rusts; general pathology.)

Chi, Chien Chen. Department of Plant Pathology, University of Wisconsin, Madison 6, Wis.

Chiarappa, Luigi. Graduate Student, University of California, Davis, Calif. (Diseases of fruit trees, grape diseases in particular.)

Childs, J. F. L. Senior Plant Pathologist, United States Department of Agriculture, Field Station, 2120 Camden

Rd., Orlando, Fla. (Virus and fungus diseases of citrus.)

Chilton, John E. Plant Pathologist, Arizona Fertilizers, Inc., P.O. Box 2191, Phoenix, Ariz. (Chemical control of vegetable crop and cotton diseases.)

Chilton, St. John P. Chairman and Professor, Department of Botany, Bacteriology, and Plant Pathology; Head, Department of Plant Pathology, Plant Pathologist, Louisiana State University, Baton Rouge, La. (Diseases of sugarcane; breeding disease-resistant varieties of sugarcane.)

Chitwood, B. G. Research Scientist, Kaiser Foundation Laboratory for Comparative Physiology and Morphology, University of California, Berkeley, Calif. (Control nemic diseases of plants; pathology, classification and physiology of Nemata, Nematomorpha, Kinorhyncha, and Tardigrada.)

Christensen, Clyde M. Professor of Plant Pathology, Department of Plant Pathology, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Industrial mycology; deterioration of stored grains by fungi.)

Christensen, Edgar V. Research Assistant, Plant Pathology Department, University of Minnesota, St. Paul 8, Minn. (Diseases of forage crops.)

Christensen, J. J. Professor and Head of the Department of Plant Pathology and Botany, 304 Plant Pathology, Institute of Agriculture, St. Paul, Minn. (Cereal diseases; genetics of fungi.)

Christiansen, Dwight W. Senior Laboratory Technician, Department of Plant Pathology, Citrus Experiment Station, University of California, Riverside, Calif. (Citrus diseases, virus and fungus.)

Christie, Jesse R. Nematologist, Agricultural Experiment Station, University of Florida, Gainesville, Fla. (Plant parasitic nematodes.)

Christou, Theodosios. Graduate Student, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Fungal diseases.)

Chun, Wallace John. Plant Quarantine Inspector, Plant Quarantine Division, P. O. Box 340, Honolulu 9, Hawaii. (Nematology.)

Chung, Hoo Sup. Instructor of Plant Pathology, College of Agriculture, Seoul University, Suwon, Korea. (Cereal diseases.)

Chupp, Charles. Professor Emeritus of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Vegetable diseases; Cercosporae.)

Ciccarone, Antonio. Professor of Plant Pathology, Istituto di Patologia vegetale, Facoltà di Agraria, Via Salerno 165/A, Bari, Italy. (Tomato, almond and olive diseases; fungus diseases of tropical and subtropical plants.)

Ciferri, Raffaele. (Life) Professor of Botany and Director of the Italian Cryptogamic Laboratory, The University, P. O. Box 165, Pavia, Italy. (Fruit tree virology; mycology; fungicides.)

Clara, Feliciano M. (Life) Bureau of Plant Industry, 692 San Andres St., Malate, Manila, Philippines.

Clarke, Oscar F. Principal Laboratory Technician, Department of Plant Nematology, University of California, Riverside, Calif. (Biological control and ecology of plant parasitic nematodes.)

Clayton, Carlyle N. Professor of Plant Pathology, Plant Pathology Department, North Carolina State College, Raleigh, N. C. (Fruit diseases.)

Clayton, Edward E. Director of Research, American Sumatra Tobacco Corporation, Mission Rd., Route 4, Box 24, Tallahassee, Fla. (Disease resistance and diseases affecting tobacco.)

Cloninger, C. Kenneth. Technical Representative, Olin Mathieson Chemical Corporation, 14 Sunset La., Columbia, Mo. (The control of plant diseases by chemicals, especially soil fungicides.)

Clum, Floyd M. Plant Pathologist, 907 Oakwood Dr., Falls Church, Va. (Host-parasite relationships; mycology.)

Clum, Harold Haydn. Professor, Department of Biological Sciences, Hunter College, 695 Park Ave., New York 21, N. Y. (Plant physiology; plant pathology.)

Cochran, George W. Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Utah State Agricultural College, Logan, Utah. (Virology; electron microscopy.)

Cochran, James H. Plant Pathologist, State Plant Board, State College, Miss.

Cochran, L. C. Horticulturist in Charge, United States Horticultural Laboratory, 2120 Camden Rd., Orlando, Fla. (Citrus production; virus diseases.)

Cochrane, Vincent W. Professor of Biology, Shanklin Laboratory, Wesleyan University, Middletown, Conn. (Physiology of fungi; parasitism.)

Cohen, Morris. Electron microscopist, Saint Joseph Hospital, Burbank, Calif. (Submicroscopic morphology; virology; proteins; cytoplasmic particulates.)

Cohen, Mortimer. Assistant Plant Pathologist, Indian River Field Laboratory, P. O. Box 1351, Fort Pierce, Fla. (Citrus diseases; virology.)

Cohen, Sylvan I. Agricultural Research Specialist, Agricultural Research Laboratory, Olin Mathieson Chemical Corporation, Box 297, Port Jefferson Station, Long Island, N. Y. (Fungicide development; tropical diseases.)

Cohill, Roger W. Miller Chemical and Fertilizer Corporation, 2226 N. Howard St., Baltimore 18, Md.

Cohoon, D. Fred. Associate Plant Pathologist, Edisto Experiment Station, Blackville, S. C. (Vegetable and fruit diseases; breeding.)

Cole, Herbert, Jr. Assistant Professor of Plant Pathology, Extension, Department of Plant Pathology, Pennsylvania State University, University Park, Pa. (General plant pathology; nematology.)

Cole, John R. Pathologist, P. O. Box 60, Albany, Ga. (Pecan diseases.)

Colley, Reginald H. Technical Director, Bernuth, Lembcke Co., Inc., 420 Lexington Ave., New York 17, N. Y. Home address for mailing: 8 Winthrop Pl., Maplewood, N. J. (Forest pathology; wood preservation; preservatives evaluation.)

Collins, Ralph P. Department of Botany, University of Connecticut, Storrs, Conn.

Comegys, Wallace R. Plant Pathologist, Rohm & Haas Company, Washington Square, Philadelphia 5, Pa. (Fungicides.)

Conant, George H. Owner, Triarch Products, Ripon, Wis. (General microtechnic; mechanics of disease resistance.)

Conners, Ibra L. Senior Mycologist, Botany and Plant Pathology Division, Science Service Building, Ottawa, Ontario, Canada. (Plant disease survey; smuts and rusts.)

Conover, Robert A. Plant Pathologist, Sub-Tropical Experiment Station, University of Florida, Route 2, Box 508, Homestead, Fla. (Vegetable diseases; fungicides.)

Converse, Richard Hugo. Plant Pathologist, Small Fruit and Grape Section, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Small fruit diseases; fungus physiology.)

Cook, Allyn A. Assistant Plant Pathologist, Department of Plant Pathology, University of Florida, Gainesville, Fla. (Vegetable diseases.)

Cook, F. E. Stauffer Chemical Company, Chauncey, N. Y. (Fungicides.)

Cook, Harold T. Agricultural Administrator, Assistant Chief, Biological Sciences Branch, Marketing Research Division, United States Department of Agriculture, Washington, D. C. (Transportation, storage, and marketing diseases of fruits and vegetables; handling of fresh food supplies for military forces.)

Cooley, Jacquelin S. (Emeritus) Collaborator, U. S. Plant Industry Station, Beltsville, Md. 10409 Montgomery Rd., Beltsville, Md.

Coons, George H. (Life) Collaborator, Tobacco and Sugar Crops Branch, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Sugar beet diseases; breeding for disease resistance.)

Cooper, Frank Edward. Graduate Student, Department of Botany, University of Maryland, College Park, Md. (Virology; tissue culture.)

Cooper, William E. Assistant Professor of Plant Pathology, North Carolina State College, Box 5397, Raleigh, N. C. (Field crop diseases; soil-borne diseases.)

Corbett, M. Kenneth. Assistant Plant Pathologist, Plant Pathology Department, University of Florida, Gainesville, Fla. (Virology.)

Corden, Malcolm E. Assistant Plant Pathologist, Crop Protection Institute, The Connecticut Agricultural Experiment Station, P. O. Box 1106, New Haven 4, Conn. (Chemotherapy; physiology of fungus parasitism.)

Corkins, Jack P. Area Supervisor, Research and Development, Naugatuck Chemical Division, United States Rubber Company, P. O. Box 6818, Los Angeles 22, Calif. (Soil fungicides.)

Cormack, M. W. Chief, Canada Agriculture Research Laboratory, University Sub Post Office, Saskatoon, Saskatchewan, Canada. (Forage crop diseases; low-temperature pathogens.)

Costa, Alvaro S. Instituto Agronomico, Campinas Est, Sao Paulo, Brazil, South America.

Cotter, Ralph U. (Life) Division of Plant Pathology, University Farm, St. Paul 8, Minn.

Couch, Houston B. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, The Pennsylvania State University, University Park, Pa. (Diseases of turf and forage crops; pathological anatomy.)

Coursen, Bradner W. Graduate Assistant, Department of Botany, University of Maryland, College Park, Md. (Fungus physiology.)

Courtney, Wilbur D. Nematologist, Section of Nematology, Crops Research Division, United States Department of Agriculture, Western Washington Experiment Station, Puyallup, Wash. (Phytonematology.)

Covey, Ronald P., Jr. Botany Department, University of Minnesota, St. Paul 1, Minn.

Cowling, Ellis B. Plant Pathologist, United States Forest Products Laboratory, Madison 5, Wis.; Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Forest products pathology; biochemistry of decay; forest pathology.)

Cox, Carroll E. Professor, Department of Botany, University of Maryland, College Park, Md. (Fungicides; fungicidal action; diseases of vegetable crops.)

Cox, Robert S. Plant Disease Consultant, 1404 Shirley Court, Lake Worth, Fla. (Vegetable pathology; fungicides.)

Coyier, Duane L. Graduate Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Diseases of ornamental plants.)

Craig, F. Waldo. Chief, Plant Pest Control, West Virginia Department of Agriculture, Capitol Building, Charleston 5, W. Va. (Regulatory.)

Craigie, J. H. Principal Plant Pathologist, Science Service Building, Carling Ave., Ottawa, Ontario, Canada. (Cereal rusts.)

Crall, James M. Plant Pathologist in Charge, Watermelon and Grape Investigations Laboratory, Florida Agricultural Experiment Station, Leesburg, Fla. (Diseases of watermelon, grape, soybean, and forage legumes; watermelon breeding.)

Cralley, E. M. Professor of Plant Pathology and Head, Department of Plant Pathology, 231 Agriculture Building, University of Arkansas, Fayetteville, Ark. (Rice diseases.)

Crandall, Bowen S. Food and Agricultural Officer, United States Operations Mission, American Embassy, Tegucigalpa, Honduras, Central America. (Root diseases.)

Creager, Don B. Chief Plant Pathologist, Florida State Plant Board Laboratory, P. O. Box 3777, University Station, Gainesville, Fla. (General plant pathology; mycology; ornamentals.)

Crispin, Alfonso. Graduate Student, Department of Plant Pathology, University of California, Davis, Calif. (Bean diseases and breeding.)

Critopoulos, Pat. D. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, University of Athens, 104 Solonos St., Athens, Greece. (Cereal rusts; Phytophthora diseases; virus diseases of grape.)

Crittenden, H. William. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of Delaware, Newark, Del. (Diseases of field crops; root pathology.)

Cromarty, Robert William. Graduate Student, Department of Plant Science, Division of Plant Pathology, University of Alberta, Edmonton, Alberta, Canada. (Plant pathology.)

Crone, Lawrence J. Plant Pathology Department, Rutgers University, New Brunswick, N. J.

Crossan, Donald Franklin. Assistant Research Professor, Department of Plant Pathology, Delaware Agricultural Experiment Station, University of Delaware, Newark, Del. (Vegetable diseases; fungicides, teaching.)

Cummings, Robert H. Plant Pathologist, Research Specialist, California Spray-Chemical Corporation, Box 129, Maryland Heights, Mo. (Fungicides.)

Cummins, George B. Professor, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Plant rust fungi.)

Cunningham, H. S. R. F. D., Calverton, N. Y.

Cunningham, John L. Graduate Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Vegetable pathology, pea root rots.)

Curl, Elroy A. Associate Plant Pathologist, Department of Botany and Plant Pathology, Alabama Polytechnic Institute, Auburn, Ala. (Soil microbiology of plant diseases; diseases of forage crops.)

Daines, Robert H. Professor and Research Specialist in Plant Pathology, Department of Plant Pathology, College of Agriculture, New Brunswick, N. J. (Diseases of tree fruits and sweet potatoes; fungicides; air pollution.)

Dale, J. L. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Corn; pasture grasses; viruses.)

Dallimore, Clarence Eugene. Assistant Plant Pathologist, Branch Experiment Station, University of Idaho, Aberdeen, Idaho. (Nematology.)

Daly, J. M. Associate Professor of Botany and Associate Plant Pathologist, University of Nebraska, Lincoln, Nebr. (Physiological and biochemical aspects of disease.)

Dance, Claude A. 1701 Fairfield Ave., Shreveport, La.

Darby, John F. Plant Pathologist, Central Florida Experiment Station, P. O. Box 327, Sanford, Fla. (Diseases of celery, crucifers, cucurbits, tomatoes, lettuce, and beans.)

Darker, Grant D. Mycologist, Ben Venue Labs., Inc., Bedford, Ohio. (Antibiotics; fungi of conifer needles.)

Darley, Ellis F. Associate Plant Pathologist, Department of Plant Pathology, University of California Citrus Experiment Station, Riverside, Calif. (Armillaria root rot; air pollution injury to vegetation; date and ornamental palm diseases.)

Darling, H. M. Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Potato diseases, nematodes.)

Darlington, Walter A. Biochemist, Monsanto Chemical Company, Dayton, Ohio. (Virology, fungicides.)

Das-Gupta, S. N. Professor and Head of the Botany Department, Department of Botany, Lucknow University, Lucknow, India. (Microbiology with special reference

to mycology and plant pathology; air pollution damage to vegetation.)

Davidson, R. S. Chief, Biosciences Division, Battelle Memorial Institute, 505 King Ave., Columbus 1, Ohio. (Fungicides; chemotherapy; nature of fungicidal action.)

Davidson, Ross W. Forest Pathologist, Forest Disease Laboratory, Plant Industry Station, Beltsville, Md. (Decay and other defects in trees and wood products.)

Davidson, Thomas Ralph. Plant Pathologist, Laboratory of Plant Pathology, Box 506, St. Catharines, Ontario, Canada. (Virology.)

Davies, Fred R. (Emeritus) Research Microbiologist in Charge of Enzyme Production, Rohm & Haas Company, Bristol, Pa. (Physiology of fungi and bacteria.)

Davis, Benjamin H. Professor of Plant Pathology, Department of Plant Pathology, Rutgers University, New Brunswick, N. J. (Diseases of vegetable crops.)

Davis, David. Plant Pathologist, Merck and Company, Inc., Rahway, N. J. (Fungicides; chemotherapy; host-parasite interactions.)

Davis, Glen N. Professor of vegetable crops and Olericulturist in the Experiment Station, Department of Vegetable Crops, University of California, Davis, Calif. (Plant breeding for disease resistance.)

Davis, Lily H. Principal Laboratory Technician, Department of Plant Pathology, University of California, 405 Hilgard Ave., Los Angeles 24, Calif. (Diseases of ornamentals; Fungi Imperfecti.)

Davis, Robert A. Graduate Student, Department of Botany & Plant Pathology, Michigan State University, East Lansing, Mich. (Vegetable diseases; fungicides.)

Davis, Ronald Allan. Graduate Assistant, Plant Pathology Department, University of Maryland, College Park, Md. (Plant nematology.)

Davis, Spencer H., Jr. Extension Specialist in Plant Pathology, Department of Plant Pathology, College of Agriculture, Rutgers University, New Brunswick, N. J. (Extension plant pathology, all phases.)

Davis, Vernon H. Horticulturist, Farm Bureau Cooperative Association, 245 N. High St., Columbus 16, Ohio. (Insecticides; fungicides, herbicides.)

Davison, Arlen D. Graduate Research Fellow, Botany and Plant Pathology Department, Oregon State College, Corvallis, Ore. (Vegetable and cereal diseases.)

Dean, Horace S. Assistant Director, Plant Quarantine Division, Agricultural Research Service, United States Department of Agriculture, Washington 25, D. C. (Plant disease prevention.)

Dean, Jack L. Associate Plant Pathologist, United States Sugar Crops Field Station, Route 6, Box 42, Meridian, Miss. (Sorghum and sugarcane diseases.)

Debbagh, Khalid Abdulkadir. Assistant Plant Pathologist, Plant Pathology Division, Abu Gurabe Experimental Farm, Abu Gurabe, Baghdad, Iraq. (Diseases of field crops.)

de Bruyn, Helen L. G. Botanist, Hinkeloordsche Weg 5, Wageningen, The Netherlands.

Decker, Phares. Professor and Head of Department of Plant Pathology, Department of Plant Pathology, Florida Agricultural Experiment Station, Gainesville, Fla. (General disease control; breeding for disease resistance.)

Deems, Robert E. Plant Pathologist, Agricultural Chemicals Section, Products Research Division, American Cyanamid Company, Stamford, Conn. (Fungicides; nematocides; general pathology.)

Deep, Ira Washington. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Teaching plant pathology; bacterial diseases; fungicide screening.)

De la Isla, Lourdes. Plant Pathologist, Investigaciones Agricolas, Laboratorio Central, Balderas 94, Mexico, D. F., Mexico. (Forage crops and strawberry nematodes.)

Delp, Charles J. Senior Research Investigator, Agricultural Chemical Section, Grasselli Chemicals Department, E. I. duPont de Nemours and Company, Wilmington, Del. (Fungicides; nematocides.)

deOng, E. R. Consultant in Agricultural Technology, 926 Stannage Ave., Albany 6, Calif. (Pesticides.)

Desai, Madhusudan V. Research Officer, P. O. Institute of Agriculture, Anand, W. Rly., India. (Seed treatments; seed rot and seeding necrosis caused by Pythium; disease control.)

Desjardins, Paul R. Assistant Plant Pathologist, Department of Plant Pathology, Citrus Experiment Station, University of California, Riverside, Calif. (Virology; diseases of citrus, avocado, and vegetable.)

Desrosiers, Russell. Plant Pathologist, Latin American Division, International Cooperation Administration, Washington 25, D. C. (Tropical plant pathology; diseases of cacao and bananas.)

DeVay, James E. Assistant Professor of Plant Pathology and Assistant Plant Pathologist in the Experiment Station, Department of Plant Pathology, University of California, Davis, Calif. (Physiology of plant pathogens and host-parasite relationships; bacterial diseases.)

deZeeuw, Donald J. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Vegetable pathology; seed treatment; diseases of cucurbits.)

Diachun, Stephen. Professor of Plant Pathology and Plant Pathologist, Kentucky Agricultural Experiment Station, Lexington, Ky. (Virus diseases of forage legumes and tobacco.)

Diaz, Jaime R. Graduate Student, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Pathology and tropical crops.)

Dickens, Lester E. Assistant Professor of Botany and Plant Pathology, Colorado State University, Fort Collins, Colo. (Tree fruit diseases.)

Dickerson, Ottie J. Private, 9766 TSU CMIC, Fort Detrick, Frederick, Md.

Dickey, Robert S. Associate Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Potato and cereal diseases.)

Dickinson, Sydney. School of Agriculture, Cambridge, England.

Dickson, B. T. (Life) Retired Chief, Division of Plant Industry, Commonwealth Scientific and Industrial Research Organization, 3 Normanby Crescent, Canberra, Australia. (Arid zone research; university education.)

Dickson, James G. Agent, Cereal Crops Section, United States Department of Agriculture; Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Field crops diseases; physiology of disease development and resistance; fungus genetics.)

Dickson, R. C. Citrus Experiment Station, Riverside Calif.

Diener, Theodor O. Associate Plant Pathologist, Irrigation Experiment Station, Prosser, Wash. (Virology; diseases of fruit crops; physiology of disease.)

Diener, Urban L. Associate Plant Pathologist, Department of Botany and Plant Pathology, Alabama Polytechnic Institute, Auburn, Ala. (Seed-borne fungi; fungicides; vegetable and fruit diseases.)

Dietz, S. M. Chairman of Botany and Plant Pathology, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Disease resistance cereal pathology; tree diseases; teaching.)

Dietz, Sherl M. Plant Pathologist, New Crops Research Branch, Agricultural Research Service, United States Department of Agriculture, Room 201, Services Building, Regional Plant Introduction Station, State College of Washington, Pullman, Wash. (Smuts; general; teaching.)

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chestnuts; reduction of wood decay in basementless houses with soil cover; sweetgum blight disease.)

DiMarco, G. R. Plant Pathology Department, Rutgers University, New Brunswick, N. J.

Dimitman, Jerome Eugene. Associate Professor of Biological Science, California State Polytechnic College, Pomona, Calif. (Citrus viruses; Phytophthora diseases.)

Dimock, A. W. Professor of Plant Pathology, Cornell University, Ithaca, N. Y. (Diseases of ornamentals.)

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Dippenaar, Barend Jacobus. Professor of Plant Pathology and Microbiology, University of Pretoria, Pretoria, Union of South Africa. (General pathology; environmental effects; deficiency diseases; mites in relation to plant diseases.)

Dobie, Norman D. Assistant Plant Pathologist, Botany and Plant Pathology Department, Cordley Hall, Room 106, Oregon State College, Corvallis, Ore. (Fungus diseases of tree fruits; fungicides; strawberry diseases.)

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Dodge, Bernard O. (Emeritus) Plant Pathologist, Emeritus, New York Botanical Garden, New York 58, N. Y. (Morphology and genetics of the fungi; plant diseases.)

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Drayton, W. W. Qualified Student, Caltex Oil (Aust.) P/L, Caltex House, Kent St., Sydney, N.S.W., Australia. (Fungicides; virology; orchard and cereal diseases; orchidology; the Thysanoptera and Homoptera.)

Drechsler, Charles. Mycologist, Plant Industry Station, Beltsville, Md. (Phycomycetes causing root rot; fungi destructive to soil animalcula; readily culturable Entomophthorales.)

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Drescher, Robert F. Research Microbiologist, Paper Section, Buckman Laboratories, Inc., 1256 North McLean Blvd., Memphis, Tenn. (Industrial microbiology.)

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DuCharme, E. P. Plant Pathologist, Citrus Experiment Station, University of Florida, Lake Alfred, Fla. (Citrus diseases.)

Duffus, James E. Plant Pathologist, United States Agricultural Research Station, Salinas, Calif. (Sugar beet diseases; virology.)

Dufrenoy, Jean. Cons. Nat'l. Des. A&M, 292 Rue St. Martin, Paris, France.

Dulaney, Eugene L. Senior Microbiologist, Research Labs., Merck, Sharp and Dohme, Rahway, N. J. (Physiology and genetics of microorganisms; industrial microbiology.)

Dunegan, John C. Retired Principal Pathologist, United States Department of Agriculture; Consultant on Special Problems, Muirkirk, Md. (Fruit diseases; fungicides; spray machinery; banana disease problems; antibiotics.)

Dunleavy, John M. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Soybean diseases.)

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Evans, Stuart. Field Representative, Arizona Fertilizers Inc., P. O. Box 2191, Phoenix, Ariz. (Lettuce and cotton diseases; fungicides; general pathology.)

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Felix, Earl Louis. (Life) Associate Plant Pathologist, Agricultural Experiment Station, University of Tennessee, Knoxville, Tenn. (Phytophthora resistance, tomato and strawberry.)

Felton, Mathias W. Plant Pathologist, Nebraska Certified Potato Growers Association, Alliance, Nebr. (Diseases of vegetable crops; production of foundation seed.)

Fenne, S. B. (Emeritus) Extension Plant Pathologist, Project Leader, Extension Service, Virginia Polytechnic Institute, Blacksburg, Va. (General plant pathology, especially tobacco diseases.)

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Fergus, Charles L. Associate Professor of Botany and Plant Pathology, Pennsylvania State University, University Park, Pa. (Taxonomy and physiology of fungus tree pathogens.)

Ferguson, John. Plant Pathologist, Soil and Plant Laboratory, Inc., Box 308, Orange, Calif. (Soil organisms; soil treatments; floricultural pathology.)

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Ferris, John M. Assistant Professor, Department of Entomology, Purdue University, Lafayette, Ind. (Plant parasitic nematodes.)

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Flor, Harold H. Plant Pathologist, State College Station, Fargo, N. Dak. (Flax diseases; genetics of host-parasite interaction.)

Flores, Marco Aurelio. Head, Division of Plant Proteccion, Ministry of Agriculture, Servicio Cooperativo Interamericano de Agricultura (SCIDA), La Aurora, Guatemala City, Guatemala, Central America. (Coffee diseases.)

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Foll, Stanley C. Research Director, H. V. Hunkel Memorial Foundation, 8951 North Green Bay Rd., Milwaukee 9, Wis. (Floricultural and nursery diseases.)

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Ford, Richard Earl. Research Assistant and Graduate Student, Plant Pathology Department, Cornell University, Ithaca, N. Y. (Forage crop diseases.)

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Franco, Emmanuel. Chefe da Inspetoria Regional de Defesa Sanitária Vegetal, Aracaju, Sergipe, Brasil. (Nematology; tropical plants diseases; fungicides.)

Frazier, Norman W. Lecturer in Entomology and Associate Entomologist, Department of Entomology, University of California, Berkeley 4, Calif. (Insect vectors of plant virus diseases, especially strawberries and grapes; Cicadellidae.)

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Friedman, William. Plant Quarantine Inspector, Plant Quarantine Division, Agricultural Research Service, United States Department of Agriculture, 904 Federal Building, 641 Washington St., New York 14, N. Y. (Plant parasitic nematodes.)

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Fulton, Neil D. Assistant Professor of Plant Pathology, Plant Pathology Department, University of Arkansas, Fayetteville, Ark. (Cotton diseases; soil microbiology.)

Fulton, Robert Harry, Assistant Professor, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Small fruit diseases; fungicides; virology.)

Fulton, Robert W. Associate Professor of Plant Pathology, Plant Pathology Department, University of Wisconsin, Madison, Wis. (Virology; virus diseases of stone fruits.)

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Garren, Kenneth H. Senior Plant Pathologist, Oilseed Branch, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Holland, Va. (Diseases of peanuts, forest pathology.)

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Gravatt, Annie R. Plant Pathologist, Crops Research Division, Plant Industry Station, Beltsville, Md. (Forest pathology; pathology of horticultural crop plants.)

Gravatt, G. Flippo. Senior Pathologist, Fruit and Nut Crops Branch, Plant Industry Station, Beltsville, Md. (Forest pathology; nut pathology, especially chestnut blight and ink disease; introduced epidemic diseases.)

Graves, Arthur H. (Life) Consultant in Genetics, The Connecticut Agricultural Experiment Station, New Haven, Conn., Wallingford, Conn. (Breeding of forest trees; diseases of trees.)

Graves, Clinton H., Jr. Assistant Plant Pathologist, Agricultural Experiment Station, Mississippi State College, State College, Miss. (Diseases of ornamental and fruit crops; fungicides.)

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Heimbeck, (Mrs.) Louise Solberg. Levanger, Norway. (L. forms of bacteria as plant pathogens.)

Heinis, Julius Leo. Plant Pathologist I, Oregon State Department of Agriculture, Salem, Ore. (Stone fruit viruses; nursery diseases.)

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Janson, Blair F. Associate Professor of Botany and Plant Pathology, Extension Plant Pathologist, Department of Botany and Plant Pathology, Ohio State University, Columbus 10, Ohio. (Plant disease control; seed treatment; virology.)

Jarvis, Karl H. Corn Breeder, Pioneer Hi-Bred Corn Company, Johnston, Iowa. (Development of new corn varieties with emphasis on disease and insect resistance.)

Jedlinski, Henry. Plant Pathology Department, University of Nebraska, Lincoln, Nebr.

Jeffers, Walter F. Plant Pathologist, Rayner Brothers, Inc., Salisbury, Md. (Diseases of strawberries and blueberries; general mycology.)

Jehle, Robert Andrew. (Life) Collaborator, Plant Disease Survey, United States Department of Agriculture; Retired State Plant Pathologist, University of Maryland, 2826 Munson St., Silver Springs, Md. (Field experiments or demonstrations for control of crop diseases; plant disease surveys.)

Jenkins, William Robert. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, University of Maryland, College Park, Md. (Plant nematology.)

Jennings, Peter R. Assistant Geneticist, Rockefeller Foundation, Apartado Aero 58-13, Bogota, Columbia, South America. (Rice breeding.)

Jensen, Dilworth D. Associate Professor of Entomology, Department of Entomology and Parasitology, University of California, Berkeley, Calif. (Insect vectors of plant viruses; viruses of orchids and of stone fruits.)

Jensen, Harold James. Associate Professor of Plant Pathology and Associate Nematologist, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Nematodes; nematode diseases; soil fumigation.)

Jensen, James H. Professor of Botany and Provost, 117 Beardshear, Iowa State College, Ames, Iowa. (Educational administration; plant virus diseases.)

Jensen, Stanley, Department of Plant Pathology, University of Nebraska, Lincoln, Nebr.

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Johannessen, George A. Division Agronomist, American Can Company, 111 Sutter St., San Francisco 4, Calif. (Vegetable and fruit diseases.)

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Johnson, F. R. Research Representative, Olin Mathieson Chemical Corporation, 419 Turner Terrace, San Mateo, Calif. (Soil-borne diseases; agricultural chemicals.)

Johnson, Folke. Plant Pathologist, Western Washington Experiment Station, Puyallup, Wash. (Diseases of fruits; air pollution.)

Johnson, Herbert G. Extension Plant Pathologist, University Farm, St. Paul 1, Minn. (Extension; plant diseases.)

Johnson, Howard B. P. O. Box 1425, Harlingen, Tex.

Johnson, Howard W. Senior Pathologist, Delta Branch Experiment Station, Crops Research Division, Agricultural Research Service, Stoneville, Miss. (Forage crops diseases.)

Johnson, Leander F. Associate Plant Pathologist, Department of Plant Pathology, University of Tennessee, Knoxville, Tenn. (Biological control; microbial antagonisms; soil-borne diseases.)

Johnson, Nels J. Dendrologist, Own business, 912 Pitner Ave., Evanston, Ill. (Forest and allied plant pathology.)

Johnson, Thorvaldur. Chief, Canada Department of Agriculture Research Laboratory, University of Manitoba, Box 322, Winnipeg, Manitoba, Canada. (Cereal diseases.)

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Johnston, (Mrs.) Peggy Stumbo. Graduate Research Assistant, Botany Department, Louisiana State University, Baton Rouge, La. (Plant pathology.)

Johnston, Titus M. Graduate Research Assistant, Botany Department, Louisiana State University, Baton Rouge, La. (Plant pathology; nematology.)

Jones, Edward D. Plant Pathologist, Red Dot Foods, Inc., Madison, Wis. (Vegetable diseases.)

Jones, John P. Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Stoneville, Miss. (Soybean diseases.)

Jones, John Paul. Graduate Student, Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Vegetable diseases.)

Jones, Laurence S. Entomologist, United States Department of Agriculture, P. O. Box 1066, Riverside, Calif. (Plant virus vectors; fruit tree viruses.)

Jones, Leo Edward. Associate Professor of Botany, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Teaching; virology; host-parasite relationships.)

Jones, Leon K. (Life) Supervisory Plant Quarantine Inspector, Plant Quarantine Division, United States Department of Agriculture, 904 Federal Office Building, Seattle 4, Wash. (Diseases of ornamentals; virology.)

Jordan, W. B. Mathieson Chemical Company, Mathieson Building, Baltimore 3, Md.

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Josephson, L. M. Agronomist, Agricultural Experiment Station, Knoxville, Tenn. (Corn breeding; cereal diseases.)

Kahn, Robert P. Plant Pathologist, United States Plant Introduction Station, Plant Quarantine Division, United States Department of Agriculture, Glenn Dale, Md. (Virology; virus diseases of fruits and ornamentals; plant quarantine.)

Kailidis, Demetris S. Graduate Student, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Fungus diseases; tree diseases.)

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Karle, Harry P. Graduate Student, Department of Plant Pathology, University of California, Davis, Calif. (Fruit tree diseases; virology; grape diseases.)

Karlovic, John K. Consulting Entomologist, Box 296, Benton Harbor, Mich. (Diseases of fruits, berries, and vegetables and their control.)

Karon, Melvin L. Director of Research, Standard Fruit Company, 944 St. Charles Ave., New Orleans, La. (Diseases of bananas, fungicides.)

Karsten, Kenneth S. Manager, Vancide Department, R. T. Vanderbilt Co., Inc., 230 Park Ave., New York 17, N. Y. (Fungicides.)

Kaufman, Donald D. Graduate Student, State Trailer Park, Kent, Ohio. (Soil microbiology.)

Kaufmann, Maurice J. Plant Pathologist, United States Department of Agriculture, Department of Plant Pathol-

ogy, University of Wisconsin, Madison, Wis. (Forage grass diseases.)

Kavanagh, Thomas. Graduate Student, Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn. (Cereal and potato diseases; genetics of the fungi.)

Kazmaier, Harold E. Principal Plant Pathologist, Bettelle Memorial Institute, 505 King Ave., Columbus, Ohio. (Pesticides.)

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Keener, Paul D. Associate Plant Pathologist, Arizona Agricultural Experiment Station; Associate Professor of Plant Pathology, College of Agriculture, University of Arizona, Tucson, Ariz. (Phytopathology; mycology; virology.)

Keil, Harry L. Plant Pathologist, Biological Laboratories, Rohm and Haas Company, Bristol, Pa. (Fungicide development; systemic fungicides; chemotherapy.)

Keitt, George W. (Life) Professor of Plant Pathology, Department of Plant Pathology, Horticulture Building, University of Wisconsin, Madison 6, Wis. (Epidemiology and control of plant diseases; nature of parasitism; genetics of phytopathogenic fungi; aerial dissemination of phytopathogens; fungicides; stone fruit virus diseases.)

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Kenaga, Clare B. Plant Pathologist, Research Laboratory, Morton Chemical Company, Woodstock, Ill. (Seed treatment; fungicides.)

Kendrick, Edgar L. Plant Pathologist, Cereal Crops Branch, Crops Research Division, United States Department of Agriculture, Regional Smut Research Laboratory, Division of Plant Pathology, Agricultural Experiment Station, Pullman, Wash. (Cereal diseases, smuts.)

Kendrick, James B., Sr. (Life) Chairman, Berkeley-Davis Department of Plant Pathology, Plant Pathologist, and Professor of Plant Pathology, Department of Plant Pathology, University of California, Davis, Calif. (Field and vegetable crop diseases.)

Kendrick, James B., Jr. Associate Plant Pathologist and Lecturer in Plant Pathology, Department of Plant Pathology, Citrus Experiment Station, University of California, Riverside, Calif. (Vegetable diseases; soil fungicides.)

KenKnight, Glenn. Plant Pathologist, United States Horticultural Field Laboratory, Fort Valley, Ga. (Stone fruit viruses.)

Kennedy, Bill W. Senior Laboratory Technician, Citrus Experiment Station, University of California, Riverside, Calif. (Field crop diseases, root rots principally.)

Kent, G. C. Professor and Head of Plant Pathology, Department of Plant Pathology, New York State College of Agriculture, Cornell University, Ithaca, N. Y. (Cereal diseases; teaching; general plant pathology.)

Kenworthy, Francis T. Plant Quarantine Inspector, Nursery Stock Section, D. C. Inspection House, Plant Quarantine Division, Agricultural Research Service, United States Department of Agriculture, Washington, D. C. (Cereal, nursery stock, and virus diseases; nonparasitic diseases.)

Keplinger, J. A., Jr. Experimentalist II, Plant Pathology Department, New York State Agricultural Experiment Station, Geneva, N. Y. (Nematology; diseases of small fruits.)

Kerling, Louise C. P. Phytopathologisch Laboratorium, Willie Commelin Scholten, 20 Javalaan, Baarn, Netherlands. (Interactions of plants with pathogens.)

Kern, Frank D. (Charter, Life) Professor of Botany and Dean, Emeritus, Graduate School, Buckhout Laboratory, Pennsylvania State University, University Park, Pa. (Uredinales.)

Kern, H. Institut für Spezielle Botanik, Eidgenössische Technische Hochschule, Universitätsstrasse 2, Zürich 6, Switzerland. (Disease mechanisms; mycology.)

Kernkamp, M. F. Assistant Director, Agricultural Experiment Station, University of Minnesota, St. Paul, Minn. (Forage crop diseases.)

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Kiesling, Richard L. Associate Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Cereal diseases.)

Kightlinger, Clifford V. (Life) Professor of Agronomy, Stockbridge Hall, University of Massachusetts, Amherst, Mass. (Plant pathology, plant breeding and agronomy of tobacco.)

Kilpatrick, R. A. Plant Pathologist, United States Department of Agriculture; Lecturer, Botany Department, University of New Hampshire, Durham, N. H. (Forage crop diseases, especially clover.)

Kim, Myong Ou. Dean, College of Agriculture, Korea University, Seoul, Korea. (Pomology.)

Kim, Woon Soo. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Virology.)

Kimble, Kenneth Alan. Principal Laboratory Technician, Department of Plant Pathology, University of California, Davis, Calif. (Diseases of vegetable crops.)

Kincaid, Randall R. Plant Pathologist, North Florida Experiment Station, Quincy, Fla. (Tobacco diseases.)

King, Claude L. Extension Plant Pathologist, Kansas State College, Manhattan, Kans. (Extension.)

King, Thomas H. Professor of Plant Pathology, Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn. (Fruit and canning crops diseases; virus diseases and principles of disease control.)

Kingsolver, Charles H. Leader, Field Crops Unit, Biological Science Branch, Agricultural Marketing Service, United States Department of Agriculture, Beltsville, Md. (Storage decline of field crops; field crop pathology.)

Kirby, Robert S. Professor Emeritus of Plant Pathology Extension, Pennsylvania State University, 251 S. Barnard St., State College, Pa. (Cereal and fruit diseases.)

Kirkpatrick, Hugh C. Plant Pathologist, Tree Fruit Experiment Station, Crops Research Branch, United States Department of Agriculture, Wenatchee, Wash. (Virology.)

Kivilaan, Aleksander. Instructor, Research, Botany and Plant Pathology Department, Michigan State University, East Lansing, Mich. (Diseases of ornamental plants; viruses.)

Klein, Harvey. Botany and Plant Pathology, Ohio State University, Columbus, Ohio.

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Kline, David M. Plant Pathologist, United States Department of Agriculture, North Carolina State College, Box 5397, Raleigh, N. C. (Cereal diseases.)

Klisiewicz, John M. Plant Pathology Department, University of Wisconsin, Madison 6, Wis.

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Klos, Edward J. Assistant Professor of Plant Pathology, Research and Extension, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Fruit diseases; fungicides; stone fruit viruses.)

Klotz, Leo J. Professor of Plant Pathology, University of California, Riverside, Calif. (Root rots; disease resistance; fruit decay control.)

Kneebone, Leon R. Associate Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, The Pennsylvania State University, University Park, Pa. (Mushroom culture and diseases; bacterial diseases of plants.)

Knorr, L. Carl. Associate Plant Pathologist, Citrus Experiment Station, University of Florida, Lake Alfred, Fla. (Pathology, botany, and cultivation of citrus; virus diseases; tropical and subtropical plant diseases; history.)

Knutson, Kenneth W. Teaching Assistant, Department of Plant Pathology, Institute of Agriculture, University of Minnesota, St. Paul, Minn. (Potato disease, epidemiology.)

Koch, Karl. Pathologist, Eastern States Farmers' Exchange, Inc., 26 Central St., West Springfield, Mass.

Koch, L. W. Head, Plant Pathology Section, Science Service Laboratory, Harrow, Ontario, Canada. (Diseases of vegetable and field crops, especially root rots.)

Koehler, Benjamin. Professor of Plant Pathology, 212 Davenport Hall, University of Illinois, Urbana, Ill. (Corn diseases.)

Koike, Hideo. Associate Pathologist, Experiment Station, Hawaiian Sugar Planters' Association, Honolulu, Hawaii. (Soil microbiology; sugarcane diseases.)

Kolbezen, Martin J. Assistant Chemist in Plant Pathology, Citrus Experiment Station, University of California, Riverside, Calif. (Residue analyses; mode of action of fungicides; decay control in stored, packaged, or in-transit produce.)

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Kommedahl, Thor. Associate Professor, Department of Plant Pathology and Botany, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Diseases of cereals and grasses.)

Kondo, William T. Research Assistant, Department of Plant Pathology, State College of Washington, Pullman, Wash. (Preservation of fungi; fungicides.)

Kontaxis, Demetrios G. Graduate Student, Plant Pathology Department, University of California, Berkeley 4, Calif. (Virology.)

Konzak, Calvin F. Associate Agronomist, Agronomy Department, State College of Washington, Pullman, Wash. (Cereal breeding, genetics, and diseases.)

Kopf, Kenneth. Ofc. of Agriculture, OEC, APO 301, c/o Postmaster, San Francisco, Calif.

Korf, Richard Paul. Associate Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Taxonomic mycology, especially Discomycetes; Ascomycete phylogeny; nomenclature.)

Kortsen, Robert A. Farm Advisor, Agricultural Extension Service, University of California, Court House, El Centro, Calif. (Diseases of vegetable and field crops.)

Krause, Robert L. Senior Biologist, Graselli Chemicals Department, duPont Experimental Station, E. I. duPont de Nemours and Company, Wilmington, Del. (Fungicides, agricultural and industrial.)

Kreitlow, Kermit W. Principal Plant Pathologist, Crops Research Division, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Forage diseases; virology; chemotherapy.)

Kreutzer, William A. Chief Plant Pathologist, Agricultural Research Division, Shell Development Company, P. O. Box 3011, Modesto, Calif. (Soil fungicides; epidemiology of root and stem diseases.)

Krueger, H. J. Room 105, Vegetable Crops Building, University of Illinois, Urbana, Ill.

Krupka, Lawrence Ronald. Graduate Research Assistant, Department of Bacteriology, Botany, and Plant Pathology, Louisiana State University, Baton Rouge, La. (Bacterial diseases; nature of disease resistance.)

Krusberg, Lorin Ronald. Graduate Assistant, North Carolina State College, Box 5397, Raleigh, N. C. (Nematode diseases of plants.)

Kuc, Joseph. Assistant Professor, Department of Biochemistry, Botany, and Plant Pathology, Purdue University, Lafayette, Ind. (Biochemistry of plant disease resistance and immunity; plant chemistry.)

Kuhn, Cedric W. Graduate Student, Botany and Plant Pathology Department, Purdue University, West Lafayette, Ind. (Virology.)

Kulik, Martin Michael. Graduate Student, Department of Plant Pathology, Louisiana State University, Baton Rouge 3, La.

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Kuntz, James E. Associate Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Forest and shade tree diseases.)

Kurtzman, Ralph H., Jr. Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Chemotherapy of virus diseases; virology; biochemistry of disease.)

Kuss, Frederick R. Plant Pathologist, Technical Representative, California Spray Chemical Corporation, Belknap Mt. Rd., Gilford, N. H. (Fruit and vegetable diseases.)

Lachmund, Harry G. Box 422, Carmel, Calif.

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Laflin, Benjamin T., Jr. Farmer, P. O. Box 757, Thermal, Calif. (Growing grapes, dates, and asparagus.)

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Lambert, Edmund B. Pathologist, Plant Industry Station, Beltsville, Md. (Mushroom culture; cereal rust epidemiology; soil mycrobiology.)

Lambrech, Joseph A. Research Center, Union Carbide Chemicals Company, South Charleston, W. Va. (Fungicides.)

Lamey, H. Arthur. Project Associate in Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Fruit diseases; forage diseases; physiology of microorganisms.)

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Langdon, Kenneth R. Graduate Student, Botany and Plant Pathology Department, Oklahoma State University, Stillwater, Okla. (Cereal diseases; fungicides.)

Langford, Arthur N. Professor of Biology, Department of Biology, Bishop's University, Lennoxville, Quebec, Canada. (Teaching of botany; breeding for disease resistance.)

Langford, Michael H. Plant Pathology Advisor, United States Operations Mission to Panama, c/o American Embassy, Panama, Republic of Panama. (Tropical plant pathology.)

Larance, Robert S. Instructor of Biology, Department of Biology, Eastern Kentucky State College, Richmond, Ky. (Botany and plant pathology.)

Large, John R. Associate Pathologist, Pecan Investigations Laboratory, Florida Experiment Station, University of Florida, P. O. Box 127, Monticello, Fla. (Fungicides; pecan, peach, and tung diseases.)

Larsen, John E. Agricultural Research Division, Shell Development Company, P. O. Box 1531, Modesto, Calif.

Larson, Russell H. Plant Pathologist, Crops Research Division, Agricultural Research Service, University of

Wisconsin, Madison 6, Wis. (Vegetable diseases, especially cabbage, onions, and potatoes; virology.)

Latch, Garrick Cecil Morland. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Forage crop diseases.)

Latham, Archie J. Graduate Student, Research Fellow, Department of Plant Pathology, College of Agriculture, University of Idaho, Moscow, Idaho. (Diseases of vegetable crops; biological control of root rot.)

Latham, Dennis H. Associate Plant Pathologist, Agricultural Experiment Station, University of Tennessee, Springfield, Tenn. (Tobacco culture and diseases.)

Latterell, Frances Meehan. Plant Pathologist, Chemical Corps Biological Laboratories, Fort Detrick, Frederick, Md. (Diseases of cereal crops; physiologic specialization.)

Lautz, William. Nematologist, Central Florida Experiment Station, Sanford, Fla. (Blackshank of tobacco; vegetable nematodes of Florida.)

Lawyer, Lewis O. 4123 Oak Hill Road, Oakland 5, Calif.

Leach, Charles M. Assistant Plant Pathologist, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Forage legume diseases and seedborne parasites.)

Leach, Julian G. Professor of Plant Pathology and Department Head, 401 Brooks Hall, West Virginia University, Morgantown, W. Va. (Plant pathology; entomology; insect transmission of plant diseases.)

Leach, Lysle Douglas. (Life) Plant Pathologist and Professor of Plant Pathology, Department of Plant Pathology, University of California, Davis, Calif. (Seedling diseases and root rots of row crops; fungicides.)

Leaphart, Charles D. Plant Pathologist, Intermountain Forest and Range Experiment Station, Inland Empire Research Center, 157 S. Howard, Spokane, Wash. (Forest diseases and their control.)

Lear, Bert. Associate Nematologist, Department of Plant Nematology, University of California, Davis, Calif. (Nematode diseases of vegetable and field crops; soil fumigation.)

Leathers, Chester R. Assistant Professor of Botany, Department of Botany, Arizona State College, Tempe, Ariz. (Mycology; cereal diseases; vegetable diseases.)

Lebeau, F. J. c/o American Embassy, Colombo, Ceylon.

Lebeau, Jack Bertram. Senior Plant Pathologist, Science Service Laboratories, Box 270, Lethbridge, Alberta, Canada. (Diseases of forage crops.)

Leben, Curt, Head, Agricultural Research Laboratories, Eli Lilly and Company, Greenfield, Ind. (Control of plant diseases with chemicals; microbiological genetics; antibiotics.)

LeClerg, E. L. Chief, Biometrical Services, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Statistics applied to biology.)

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Lee, Sung Woo. Dean, College of Agriculture, Tongkuk University, Seoul, Korea. (Pomology.)

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Lehman, Samuel G. Emeritus Professor of Plant Pathology, North Carolina State College, 123 Brooks Ave., Raleigh, N. C. (Diseases of soybeans and cotton.)

Le Roux, Pieter Meyer. Senior Lecturer of Plant Pathology, Agricultural Research Institute, University of Pretoria, Pretoria, Union of South Africa. (Cereal diseases.)

LeTourneau, Duane J. Assistant Professor and Assistant Agricultural Chemist, Department of Agricultural Chemistry, University of Idaho, Moscow, Idaho. (Biochemistry of fungi; biochemistry of diseased plants.)

Leukel, R. W. Bureau of Plant Industry Station, Beltsville, Md.

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Lewis, Gwynne David. Graduate Student, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Vegetable diseases; nematology.)

Lewis, Ralph William. Professor of Biology, Department of Natural Science, Michigan State University, East Lansing, Mich. (Host-parasite relationship; nutrition.)

Leyendecker, Philip J., Jr. Head, Department of Agricultural Services, New Mexico College of Agriculture and Mechanic Arts, State College, N. Mex. (Verticillium wilt; disease loss surveys; cotton diseases.)

Libby, Robert R. Plant Pathologist, Stauffer Chemical Company, 380 Madison Ave., New York 17, N. Y. (Fungicides.)

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Liming, O. Neal. Deputy Director, Department of Agriculture, Government of Guam, Agana, Guam. (All aspects of agriculture.)

Lindaberry, Harold. Technical Supervisor, Pennsalt Chemicals, 309 Graham Building, Aurora, Ill. (Technical service; field evaluations, formulation; etc.)

Lindberg, George D. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, Louisiana State University, Baton Rouge, La. (Forage crops; rice; virology.)

Lindgren, Ralph M. Chief, Division of Wood Preservation, United States Forest Products Laboratory, Madison, Wis. (Tree and wood products pathology; microbiology; fungus control.)

Lindner, Robert C. Plant Pathologist, Tree Fruit Experiment Station, Route No. 4, Wenatchee, Wash. (Virology.)

Line, Roland F. Research Assistant, Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn. (Plant pathology; plant genetics.)

Linford, Maurice B. (Life) Professor of Plant Pathology, Plant Pathology Department, University of Illinois, Urbana, Ill. (Plant nematology.)

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Lippert, Laverne F. Graduate Student, Department of Vegetable Crops, University of California, Davis, Calif. (Vegetable crops diseases.)

Litzenberger, Samuel C. Agriculturist-Research Advisor, International Cooperation Administration, United States Operations Mission to Cambodia, c/o American Embassy, Phnom Penh, Cambodia. (Plant disease survey and con-

trol, during recent years in sub-tropical and tropical environments.)

Liu, Stephen Chieh-yuan. Research Fellow, Department of Plant Pathology, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Virology; physiology of plant pathogens; plant virus serology.)

Livingston, Clark H. Assistant Professor of Botany and Plant Pathology, Botany Department, Colorado State University, Fort Collins, Colo. (Potato pathology; potato virology; physiology of disease.)

Livingston, J. E. Head, Department of Botany and Plant Pathology, 215 Deering Hall, University of Maine, Orono, Maine. (Chemotherapy; potato and cereal diseases.)

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Lockwood, John L. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Vegetable diseases; antibiotics and antagonism.)

Loegering, William Querin. Senior Plant Pathologist, Crops Research Division, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Genetics of stem rust organism; isolation of genes in wheat for resistance to stem rust; diseases of abaca.)

Logsdon, Charles E. Plant Pathologist, Alaska Agricultural Experiment Station, Palmer, Alaska. (Potato diseases; cereal and forage diseases.)

Lohman, Marion L. 326 Illinois Ave., Dayton 10, Ohio. (General; applied mycology in particular.)

Long, William G. Horticulturist, Quality Maintenance and Improvement Section, Agricultural Marketing Service, United States Department of Agriculture, 2120 Camden Rd., Orlando, Fla. (Disease control and epiphytology; diseases of citrus fruit and vegetables, including market disease.)

Loos, Clive A. Plant Pathologist, Research Laboratory, Chiriqui Land Company, Almirante, Republic of Panama. (Nematology; banana diseases.)

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Loring, Lindsay B. Plant Pathologist, State Department of Agriculture, Salem, Ore. (Disease survey; control of diseases of ornamentals; nematology.)

Lownsbery, Benjamin Ferris. Assistant Nematologist, Department of Plant Nematology, University of California, Davis, Calif. (Physiology and control of plant parasitic nematodes; etiology and pathogenesis of disease involving plant parasitic nematodes.)

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Luepschen, Norman S. Graduate Research Assistant, Plant Pathology Department, Cornell University, Ithaca, N. Y. (Diseases of tree fruits; antibiotics; spray machinery.)

Luke, H. H. Plant Pathologist, Cereal Crops Research Branch, United States Department of Agriculture, Plant Pathology Department, University of Florida, Gainesville, Fla. (Small grain diseases.)

Lukens, Raymond J. Assistant Plant Pathologist, Connecticut Agricultural Experiment Station, 123 Huntington Ave., New Haven, Conn. (Fungicides and their action; diseases of nursery plants.)

Lukezic, Felix L. Graduate Student, Botany and Plant Pathology Department, Colorado State University, Fort Collins, Colo. (Virology.)

Lutes, Dallas D. Associate Professor of Botany and Bacteriology, Louisiana Polytechnic Institute, Ruston, La. (Teaching; general pathological research.)

Lutey, Richard W. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Luttrell, E. S. Plant Pathologist, Georgia Experiment Station, Experiment, Ga. (Morphology and taxonomy of Ascomycetes and Fungi Imperfecti; diseases of forage crops.)

Lyda, Stuart D. Graduate Student, Botany and Bacteriology Department, Montana State College, Bozeman, Mont. (Fungus diseases of plants.)

Lyerly, Paul J. Superintendent, Substation 17, Texas Agricultural Experiment Station, Route 1, Box 454, Ysleta, Tex. (Cotton diseases.)

Lyle, Eldon W. Plant Pathologist, Texas Rose Research Foundation, Inc., Route 9, Box 105, Tyler, Tex. (Roses.)

Lyle, James A. Head of Department and Head Professor, Botany and Plant Pathology Department, Alabama Polytechnic Institute, Auburn, Ala. (Peanut, cereal, and grass diseases.)

McAnelly, Charles W. Department of Agronomy, University of Wyoming, Laramie, Wyo.

McCain, Arthur H. Graduate Student in Plant Pathology, Department of Plant Pathology, University of California, Davis, Calif. (General plant pathology.)

McCallan, S. E. A. Plant Pathologist, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Fungicides.)

MacCarthy, Hubert R. Head, Crop Insect Laboratory, Canada Department of Agriculture Research Laboratory, c/o University of British Columbia, Vancouver 8, B. C., Canada. (Virus transmission by insects.)

McClain, R. L. Associate Plant Pathologist, Bureau of Plant Pathology, California Department of Agriculture, P. O. Box 1600, Riverside, Calif. (Citrus diseases.)

McClellan, W. D. Principal Plant Pathologist, State Experiment Stations Division, Agricultural Research Service, United States Department of Agriculture, Washington 25, D. C. (General plant pathology; experiment station administration.)

McClintock, James A. Professor of Horticulture, Purdue University, West Lafayette, Ind. (Horticulture; plant pathology.)

McClure, Thomas T. Associate Plant Pathologist, Quality Maintenance and Improvement Section, Agricultural Marketing Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Fungus diseases of fruits and vegetables; mechanisms of resistance.)

McColloch, Lacy Porter. Plant Pathologist, Biological Sciences Branch, Agricultural Marketing Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Post-harvest diseases of fruits and vegetables; relation of environment to disease occurrence and control.)

McCormick, William H. Research Assistant, Department of Plant Pathology, University of Arizona, Tucson, Ariz. (Fungal diseases; physiology of fungi.)

McCrum, Richard C. Assistant Plant Pathologist, Maine Agricultural Experiment Station, University of Maine, Orono, Maine. (Virology; forest and fruit trees.)

Macdonald, Kenneth H. Graduate Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y.

(Plant parasitic nematodes; potato diseases; disease control.)

McDonald, W. C. Plant Pathologist, Plant Pathology Section, Canada Department of Agriculture Research Laboratory, University of Manitoba, Box 322, Winnipeg, Manitoba, Canada. (Forage crop diseases.)

McDonough, E. S. Professor, Biology Department, Marquette University, Milwaukee 3, Wis.

McFadden, Lorne Austin. Assistant Horticulturist, Sub-Tropical Experiment Station, Box 508, Route 2, Homestead, Fla. (Diseases of ornamental plants.)

McFarlane, John S. Senior Geneticist, Alisal Branch, United States Agricultural Experiment Station, P. O. Box 98, Salinas, Calif. (Breeding for disease resistance.)

McGlohon, Norman Edward. Graduate Student, Botany Department, Clemson College, Clemson, S. C. (Phytonematology.)

McGrew, John R. Plant Pathologist, Small Fruit and Grape Section, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Diseases of strawberries and grapes.)

McGuire, James M. Department of Plant Pathology, North Carolina State College, Raleigh, N. C.

McIntosh, David L. Plant Pathologist, Plant Pathology Laboratory, Summerland, B. C., Canada. (Tree-fruit parasitic diseases.)

McKeen, Colin D. Senior Plant Pathologist, Science Service Laboratory, Harrow, Ontario, Canada. (Diseases of vegetable crops; virology.)

McKeen, W. E. Associate Professor of Botany, Botany Department, University of Western Ontario, London, Ontario, Canada. (Fruit and root-rot diseases; mycology; teaching.)

McKelvey, John J., Jr. Assistant Director for Agriculture, The Rockfeller Foundation, 49 West 49th St., New York 20, N. Y. (General plant pathology; economic entomology.)

McKenzie, Malcolm A. Director of Shade Tree Laboratories, University of Massachusetts, Amherst, Mass. (Diseases of ornamental, shade, and forest trees.)

McKinney, H. H. (Life) Senior Pathologist, Crops Research Division, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Virus diseases; virology.)

McLean, Darrell M. Associate Plant Pathologist, Crops Research Division, United States Department of Agriculture, Cooperating Northwestern Washington Experiment Station, Mount Vernon, Wash. (Vegetable seed crops.)

McLean, John G. Senior Horticulturist, Vegetable and Ornamental Research Branch, Crops Research Division, United States Department of Agriculture, United States-Colorado Potato Field Station, P. O. Box 747, Greeley, Colo. (Potato breeding for horticultural characters and disease resistance to Verticillium wilt, scab, leafroll, X and A viruses; biochemical resistance.)

McManus, Peter J. Manager, Pesticide Department, G.L.F. Soil Building Division, Terrace Hill, Ithaca, N. Y. (Distribution of pesticides.)

McMeekin, Dorothy. Graduate Student, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Teaching.)

MacMillan, Howard Gove. (Life) 1725 Fifth Ave., Los Angeles 19, Calif. (Native plants, their diseases and climatological relationships.)

McNabb, Harold S., Jr. Associate Professor of Forest Pathology, Departments of Botany and Plant Pathology, and Forestry, Iowa State College, Ames, Iowa. (Diseases of forest and shade trees; wood decay; the genus Quercus.)

MacNeill, Blair H. Professor of Plant Pathology, Department of Botany, Ontario Agricultural College, Guelph, Ontario, Canada. (Vegetable pathology; virology.)

McNew, George L. Managing Director, Boyce Thompson Institute for Plant Research, 1086 North Broadway, Yonkers, N. Y. (Agricultural chemicals; fungicides; parasitism.)

McRitchie, John J. Graduate Student, Department of Botany and Plant Pathology, Ohio State University, Columbus 10, Ohio. (Virology, vegetable diseases.)

MacSwan, Iain C. Assistant Professor, Plant Pathology Extension, Cordley Hall, Oregon State College, Corvallis, Ore. (Horticultural crops.)

McVey, Donald V. Graduate Student, 244 Davenport Hall, University of Illinois, Urbana, Ill. (Forage crops.)

McWhorter, Frank P. Professor of Plant Pathology, Oregon State College, Corvallis, Ore. (Ornamentals; general virology.)

Mabry, Paul H. Manager, Agricultural Chemicals Division, The Kilgore Seed Company, Plant City, Fla. (Diseases of horticultural plants; fungicides.)

Mace, Marshall Ellis. Graduate Student, Department of Plant Pathology, North Carolina State College, Raleigh, N. C. (Cereal diseases; virology.)

Machacek, John E. Senior Plant Pathologist, Canada Department of Agriculture Research Laboratory, University of Manitoba, P. O. Box 322, Winnipeg 9, Manitoba, Canada. (Seed-borne diseases of cereals and flax and their control by seed treatment.)

Machmer, John H. Nematologist, Nematology Section, Crops Research Division, United States Department of Agriculture, Texas Agricultural and Mechanical Experiment Station, Weslaco, Tex. (Plant nematology.)

Mader, E. O. 1840 Berkeley Ave., St. Paul 5, Minn. (Physiology of fungi.)

Magie, Robert O. Professor of Plant Pathology, Manatee Station, Gulf Coast Experiment Station, P. O. Box 2125, Bradenton, Fla. (Pathology of cut-flower crops; systemic fungicides.)

Mahoney, Charles H. Director, Raw Products Research Bureau, National Canners Association, 1133 20th St., N. W., Washington 6, D. C. (Fungicides; fruit and vegetable diseases.)

Mai, William F. Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Nematology; diseases of roots; nematocides.)

Maier, Charles R. Research Fellow, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Plant pathology; plant physiology; lower plants; mycology; philosophy.)

Maine, Ellsworth C. Graduate Student, Department of Plant Pathology, North Carolina State College, Raleigh, N. C. (Biochemical nature of plant disease resistance; bacterial diseases of plants.)

Mains, E. B. (Life) Professor of Botany and Director of the Herbarium, Museums Building, University of Michigan, Ann Arbor, Mich. (Entomogenous fungi; Uredinales; host specialization of rusts and powdery mildews.)

Makila, Armas A. Senior Laboratory Technician, Citrus Experiment Station, University of California, Riverside, Calif. (Vegetable diseases; mycology.)

Malaguti, Gino. Plant Pathologist, Centro de Investigaciones Agronomicas, El Limón, Maracay, Venezuela. (Rice and tropical crops diseases.)

Malca, Isaac. Graduate Student, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Physiology of fungi; diseases of field crops.)

Maloy, Otis C., Jr. Graduate Student, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Root rots; ecology and physiology of soil microorganisms.)

Mankau, Reinhold. Assistant Nematologist, Department of Plant Nematology, Citrus Experiment Station, University of California, Riverside, Calif. (Plant parasitic nematodes; biological control.)

Mankin, Cleon J. Associate Plant Pathologist, Plant Pathology Department, South Dakota State College,

Brookings, S. Dak. (Diseases of forage crops; vegetable diseases.)

Manzer, Franklin E. Graduate Assistant, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Vegetable diseases.)

Maramorosch, Karl. Assistant Professor, Rockefeller Institute for Medical Research, 66th St. and York Ave., New York 21, N. Y. (Viruses and virus diseases; insect transmission.

Marchetti, Marco Anthony. Graduate Assistant, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Oak Wilt; Plant pathology.)

Marlatt, Robert Bruce. Associate Plant Pathologist, Agricultural Experiment Station, University of Arizona, P. O. Box 925, Mesa, Ariz. (Vegetable diseases.)

Marsden, David H. Plant Pathologist, Eastern States Farmers' Exchange, Inc., 26 Central St., West Springfield, Mass. (Fungicides.)

Marshall, Norton L. Plant Pathologist, Research Department, Firestone Plantations, Harbel, Liberia, Africa. (Diseases of tropical crops; fungus physiology; fungicides.)

Marshall, Rush P. Director, Bartlett Tree Research Laboratories, 2770 Summer St., Stamford, Conn. (Disorders of shade trees.)

Martin, George H., II. Phytopathologist, Boston Post Road, South Sudbury, Mass. (Diseases of forest trees and ornamental plants.)

Martin, Helen. Biologist, Rohm and Haas Company, Bristol, Pa. (Fungicides.)

Martin, James P., Jr. Graduate Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison 6, Wi . (Forage crops.)

Martin, Weston J. Plant Pathologist, Department of Plant Pathology, Louisiana State University, Baton Rouge 3, La. (Sweet potato diseases, including nematode diseases.)

Martin, William H. (Life) Dean and Director, College of Agriculture and Agricultural Experiment Station, Rutgers, The State University, New Brunswick, N. J. (Agriculture; education.)

Martinson, Charlie A. Graduate Assistant, Department of Botany and Plant Pathology, Colorado State University, Ft. Collins, Colo. (Root-rot diseases.)

Marx, Gerald A. Graduate Student and Research Assistant, Department of Agronomy, College of Agriculture, University of Wisconsin, Madison 6, Wis. (Breeding, diseases, and physiology of canning crops, especially peas.)

Mason, Curtis L. Manager of Technical Service, Niagara Chemical Division, Food Machinery and Chemical Corporation, Middleport, N. Y. (Fungicides; fruit diseases; cotton diseases.)

Massey, Louis M. (Life) Professor of Plant Pathology, Department of Plant Pathology, The New York State College of Agriculture, Cornell University, Ithaca, N| Y. (Diseases of ornamentals; fungicides; physiology of parasitism.)

Mastenbroek, Cornelis. Plant Breeding Station, Centraal Bureau, Hoofddorp, The Netherlands. (Plant breeding, especially potatoes for disease resistance.)

Matsumoto, Takashi. (Emeritus) Professor of Plant Pathology, Phytopathological Laboratory, National Taiwan University, Taipei, Formosa, China. (Virology; tropical phytopathology.)

Mattei, John A. Plant Pathologist, F. H. Woodruff Seed Company, Division of Asgro, 2000 Pennsylvania Court, West Sacramento, Calif. (Vegetable crop diseases; soil-borne diseases; fungicides.)

Matz, Julius. R. F. D. 1, Boxford, Mass. (Tropical plant pathology.)

Maung, Ohn. Graduate Student, Department of Botany, University of Maryland, College Park, Md. (Nematode diseases.)

Maxwell, Lewis S. Entomologist, Jackson Grain Company, P. O. Box 1290, Tampa, Fla. (Insects and diseases attacking turf and ornamentals.)

May, Curtis. Principal Pathologist, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Tree diseases; systemics.)

Mehrlich, F. P. Research and Development Department, General Foods Corporation, 250 North St., White Plains, N. Y.

Meier, Mrs. Fred C. (Life) 7102 Beechwood Dr., Chevy Chase 15, Md.

Meiners, Jack P. Assistant Chief, Cereal Crops Research Branch, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Md. (Cereal diseases.)

Melander, Leonard W. (Life) Research Development, 1439 Hythe St., St. Paul 8, Minn. (Use of herbicides for eradication of weeds and woody plants and alternate hosts.)

Melchers, Leo E. Professor Emeritus of Botany and Plant Pathology, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Cereal diseases; breeding for disease resistance.)

Melching, Joseph Stanley. Graduate Research Assistant, Plant Pathology Department, Cornell University, Ithaca, N. Y. (Fungicides; physiology of parasitism.)

Melhus, Irving E. (Life) Emeritus Professor of Plant Pathology, Iowa State College, 407 Pearson Ave., Ames Iowa. (Phycomycetes; parasitism; plant exploration and variability.)

Menon, S. K. Ananda Vilas, Punkunnam, P. O. Trichur, T-C., State, India.

Menzies, James D. Microbiologist, United States Department of Agriculture, Irrigation Experiment Station, Prosser, Wash. (Soil-borne plant pathogens.)

Meuli, Lloyd J. Plant Pathologist, The Dow Chemical Company, Box 245, Seal Beach, Calif. (Soil fungicides.)

Meyer, Bernard S. Chairman, Department of Botany Plant Pathology, Ohio Agricultural Experiment Station, Ohio State University, Columbus 10, Ohio. (Plant physiology.)

Michaelson, Merle E. Plant Pathologist, United States Department of Agriculture, Department of Plant Pathology, South Dakota State College, Brookings, S. Dak. (Diseases of flax and cereals.)

Middleton, John T. Professor of Plant Pathology, Department of Plant Pathology, University of California, Riverside, Calif. (Vegetable diseases; Pythium root rots; air pollution.)

Mielke, James L. Forest Pathologist, Intermountain Forest and Range Experiment Station, Utah State Agricultural College, Logan, Utah. (Forest pathology.)

Milbrath, David Gallus. Retired Chief of Bureau of Plant Pathology, California State Department of Agriculture, 1410 41 St., Sacramento 19, Calif. (Ecology in plant diseases.)

Milbrath, John A. Plant Pathologist, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Virology; virus diseases of deciduous tree fruits; teaching graduate course in plant viruses.)

Milholland, Robert D. Botany and Plant Pathology Department, Oklahoma State University, Stillwater Okla.

Millar, Paul H. Chief Inspector, Arkansas State Plant Board, Box 1069, Little Rock, Ark. (Regulatory plant pest control.)

Miller, Harold J. Supervisor, Agricultural Chemicals Section, Pennsalt of Washington Division, 2901 Taylor Way, Tacoma 2, Wash. (Fungicide screening, formulation, and development.)

Miller, Howard N. Plant Pathologist, Plant Pathology Department, Agricultural Experiment Station, University of Florida, Gainesville, Fla. (Diseases of ornamentals.)

Miller, James D. Plant Pathologist, United States Department of Agriculture; Graduate Student, Plant

Pathology Building, University of Minnesota, St. Paul 1, Minn. (Cereal diseases.)

Miller, Joseph Austin. M. D. 1 South Centre St., South Orange, N. J.

Miller, Julian H. Chairman of Division of Plant Pathology, Department of Plant Pathology, College of Agriculture, University of Georgia, Athens, Ga. (Mycology.)

Miller, Lawrence I. Plant Pathologist, Tidewater Research Station, Holland, Va. (Nematodes; peanut diseases.)

Miller, Lawrence P. Biochemist, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Mechanisms of fungicidal action; radioisotope techniques applied to fungicide studies; fungus physiology.)

Miller, Patrick Martin. Assistant Plant Pathologist, The Connecticut Agricultural Experiment Station, 123 Huntington St., New Haven, Conn. (Nematology; fruit diseases; fungicides; soil treatments; soil-borne diseases.)

Miller, Paul R. Principal Pathologist, Mycology and Disease Reporting Section, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Disease surveys and forecasting.)

Miller, Paul William. Plant Pathologist, 120 Cordley Hall, Oregon State College, Corvallis, Ore. (Virology, strawberry virus diseases; nut diseases.)

Miller, Roy E. Miller Products Company, Foot of S. W. Caruthers St., Portland 1, Ore.

Miller, William E. Plant Pathologist, Crops Division, Fort Detrick, Frederick, Md. (Cereal pathology.)

Miller, William Harry. Graduate Teaching Assistant, Department of Botany, University of Maryland, College Park, Md. (Virology.)

Millikan, D. F. Plant Pathologist, Department of Horticulture, Room 7, Whitten Hall, University of Missouri, Columbia, Mo. (Virus diseases; virology; diseases of fruits, vegetables, and nursery crops.)

Mills, Wilford R. Professor of Plant Pathology, Department of Botany and Plant Pathology, Pennsylvania State University, University Park, Pa. (Breeding potatoes for disease resistance; races and pathogenicity of Phytophthora infestans.)

Minnum, Edward. Associate Professor of Vegetable Gardening, University of Connecticut, Storrs, Conn. (Vegetables.)

Minz, Gershon. Plant Pathologist, Division of Plant Pathology, Agricultural Research Station, Rehovot, Israel. (Field crop diseases; nematodes.)

Mirocha, Chester Joseph. Graduate Student, Senior Laboratory Technician, Department of Plant Pathology, University of California, Davis, Calif. (Fruit and nut crops.)

Mishra, Jai Narayan. Bihar Agricultural College, Sabour, Bhagalpur, India.

Mitchell, John E. Associate Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Physiology of fungicides; Physiology of plant diseases.)

Mitchell, John W. Principal Physiologist, Crops Research Division, Plant Industry Station, Beltsville, Md. (Growth regulators and antibiotics.)

Mohamed, Hosni Abdel Rahman. Plant Pathologist, Bahteem Experiment Station, Egyptian Agricultural Organization, Shoubra ElKheima P. O., Cairo, Egypt. (Cereal and cotton diseases.)

Monroe, Carl M. Plant Pathologist, Shell Development Company, P. O. Box 3011, Modesto, Calif. (Fungicide and bactericide development; epidemiology of diseases.)

Montie, Thomas C. Graduate Student, Department of Plant Pathology, University of Maryland, College Park, Md. (Antibiotics from Streptomyces; fungus physiology.)

Mook, Paul V. Plant Pathologist, New Haven Forest, Insect, and Disease Laboratory, Northeastern Forest Experiment Station, 335 Prospect St., New Haven 11, Conn. (Forest tree diseases.)

Moore, Bernard J. Graduate Student, 231 Agriculture Building, University of Arkansas, Fayetteville, Ark. (Diseases of cotton seedlings.)

Moore, Elizabeth J. Medical Research Specialist, Louisiana State University School of Medicine, 1542 Tulane Ave., New Orleans, La. (Organic phosphate poisons; histochemistry.)

Moore, Ermer Leon. Senior Agronomist, United States Department of Agriculture; Research Associate Professor of Plant Pathology, North Carolina State College, Tobacco Research Station, Oxford, N. C. (Tobacco diseases and tobacco breeding.)

Moore, J. Duain. Agent, Plant Pathologist, Crops Research Division, United States Department of Agriculture; Professor of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Stone fruit virus diseases; fruit diseases; fungicides.)

Moore, M. B. Division of Plant Pathology, University Farm, St. Paul 1, Minn.

Moore, William D. (Life) Senior Pathologist, Florida Agricultural Experiment Station, 5305 S. W. 12 St., Fort Lauderdale, Fla. (Vegetable crop diseases.)

Moorhead, Ellen L. Plant Pathologist, Agricultural Research Service, United States Department of Agriculture, Department of Plant Pathology, University of Nebraska, Lincoln, Nebr. (Virus diseases of cereals; serological studies.)

Morgan, Omar D., Jr. Assistant Professor of Plant Pathology, Botany Department, University of Maryland, College Park, Md. (Tobacco pathology; fungicide research; nematode control.)

Morrison, Lou S. Assistant Professor, Botany and Plant Pathology Department, Oklahoma State University, Stillwater, Okla. (Fruit and vegetable pathology; nematology.)

Morton, Donald J. Assistant Plant Pathologist, New Mexico Agricultural and Mechanical State College, Box 576, N. Mex. (Plant-parasitic nematodes; viruses; cotton.)

Moseman, John G. Supervisory Plant Pathologist, Crops Research Division, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Diseases of cereal crops; genetics of the fungi; virology.)

Mountain, William B. Plant Pathologist, Science Service Laboratory, Harrow, Ontario, Canada. (Plant nematology).

Mowry, James B. Associate Professor of Horticulture and Superintendent, Illinois Horticultural Experiment Station, Southern Illinois University, Carbondale Ill. (Breeding fruits for resistance to diseases.)

Mueller, Walter C. Plant Pathology Department, Cornell University, Ithaca, N. Y.

Muir, William H. Assistant Professor of Biology, Department of Biology, Carleton College, Northfield, Minn. (Tissue culture; abnormal growth.)

Muller, Albert Stanley. Professor of Plant Pathology, McCarty Hall 117, University of Florida, Gainesville, Fla. (Plant disease surveys; mycology.)

Müller, Karl Otto Albert. (Professor of Plant Pathology, Universities Berlin and Halle, S. Germany.) Until June, 1958, Senior Research Fellow, C.S.T.R.O., 15 Clianthus St., O'Connor, Canberra, Australia. (Host-pathogen relationships, hypersensitive reaction; plant breeding for disease resistance.)

Mullin, Robert S. Plant Pathologist, Virginia Truck Experiment Station, P. O. Box 2160, Norfolk, Va. (Diseases of vegetable crops.)

Mumford, David Louis. Graduate Student, Department of Plant Pathology and Botany, University of Minnesota, St. Paul 1, Minn. (Diseases of forage crops.)

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Munger, G. Donald. Senior Plant Pathologist, Diamond Alkali Company, 8902 Michaux Lane, Richmond 26, Va. (Agricultural chemicals.)

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Munnecke, Donald E. Assistant Professor and Assistant Plant Pathologist, Department of Plant Pathology, University of California, Los Angeles 24, Calif. (Soil fungicides; diseases of ornamental crop plants.)

Munro, James. Plant Pathologist, Laboratory of Plant Pathology, Canada Department of Agriculture, Fredericton, New Brunswick, Canada. (Virology.)

Murakishi, Harry H. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Vegetable diseases; virology.)

Murphy, Albert M. Plant Pathologist, Sugar Section, Tobacco and Sugar Beet Research Division, United States Department of Agriculture, P. O. Box 826, Twin Falls, Idaho. (Sugar beet diseases; virology.)

Murphy, Hickman C. (Life) Head, Oat Section, Cereal Crops Research Branch, Crops Research Division, Beltsville, Md. (Cereal diseases.)

Nabi, Ghulam. Undergraduate Student, c/o Mr. Arnold, College of Agriculture, University of Wyoming, Laramie, Wyo. (Field crop diseases.)

Nadakavukaren, M. J. Botany and Plant Pathology Department, Oregon State College, Corvallis, Ore.

Nagel, Clatus Martin. Head, Department of Plant Pathology, Plant Pathology Building, South Dakota State College, Brookings, S. Dak. (Corn diseases; root rot diseases; tree diseases.)

Nair, P. Narayanan. Chempakamangalam, Kunnukuzhy, Trivandrum, India.

Nakayama, Takao. Agriculturist, Chofu Hydroponic Farm, United States Army Quartermaster Center at Tokyo, A.P.O. 500, San Francisco, Calif. (Vegetable diseases.)

Nakayama, Roy M. Botany and Plant Pathology Department, Iowa State College, Ames, Iowa.

Nance, Nellie W. Assistant Pathologist, Mycology and Plant Disease Reporting Section, Crops Protection Research Branch, Crops Research Division, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Plant disease reporting.)

Narvaez-Morales, Ignacio. Londres No. 40, Mexico 6, D. F., Mexico. (Wheat breeding and diseases.)

Natour, Rashad M. Graduate Student, Rolfs Hall, Agricultural Experiment Station, University of Florida, Gainesville, Fla. (Vegetable and cereal diseases.)

Natti, John J. Associate Professor of Plant Pathology, New York State Agricultural Experiment Station, Cornell University, Geneva, N. Y. (Bacterial, fungus, and virus diseases of table beets, beans, crucifers, and sweet corn.)

Nayudu, M. Venkata Rama. Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Plant pathology; plant physiology; plant biochemistry.)

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Neergaard, Paul. Statens Plantetilsyn, Gersonsvej 13, Hellerup, Denmark.

Nelson, Irvin H. Instructor of Biology, Arlington State College, Arlington, Tex. (Biological control; soil microbiology; growth regulators.)

Nelson, Klayton Edward. Assistant Viticulturist, Department of Viticulture, Davis, Calif. (Post-harvest physiology of fruit and decay organisms.)

Nelson, Merritt R. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Vegetable diseases.)

Nelson, Paul E. Assistant Professor of Plant Pathology, Ornamentals Research Laboratory, Cornell University, Farmingdale, Long Island, N. Y. (Diseases of ornamentals; root diseases; pathological anatomy.)

Nelson, Ray. Research Professor in Plant Pathology, 446 National Science Building, Michigan State University, East Lansing, Mich. (Diseases of celery, onion, mint, and gladiolus.)

Nelson, Richard R. Plant Pathologist, Crops Research Division, United States Department of Agriculture, Department of Plant Pathology, North Carolina State College, Raleigh, N. C. (Fungus genetics; nematodes; corn diseases.)

Nesbitt, Robert B., Jr. Deputy Agricultural Commissioner and County Plant Pathologist, Orange County Department of Agriculture, 9846 South Harbor Blvd., Anaheim, Calif. (General plant pathology and nematology.)

Nesom, Rivers Y. Professor of Biology and Head of the Department of Biological Sciences, Southeastern Louisiana College, Hammond, La. (General plant pathology; teaching.)

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Newhall, Allan Goodrich. (Patron) Professor of Plant Pathology, New York State College of Agriculture, Cornell University, Ithaca, N. Y. (Fungicides; soil fumigants and other treatments; diseases of tropical plants; nematodes.)

Newton, William. Officer in Charge, Plant Pathology Laboratory, Saanichton, B. C., Canada. (General plant pathology.)

Nichols, Carl W. Associate Plant Pathologist, Bureau of Plant Pathology, California State Department of Agriculture, Sacramento 14, Calif. (New disease detection; stone fruit viruses.)

Nichols, Courtland G. Graduate Student, Department of Horticulture, University of Wisconsin, Madison 6, Wis. (Breeding of vegetable crops for disease resistance.)

Nichols, Lester P. Associate Professor of Plant Pathology, Extension, 211 Botany Building, The Pennsylvania State University, University Park, Pa. (Ornamentals; trees.)

Niederhauser, John S. Plant Pathologist, Rockefeller Foundation, Londres 40, Mexico 6, D. F., Mexico. (Diseases of potatoes, corn, beans; general mycology.)

Nielsen, L. W. Professor of Plant Pathology, Division of Plant Pathology, North Carolina State College, Raleigh, N. C. (Irish potato diseases; sweet potato diseases.)

Nikitin, Alexander A. Director of Agricultural Research, Tennessee Corporation Research Laboratories, 900 Roosevelt Highway, College Park, Ga. (Plant nutrient elements; fungicides; plant life in general.)

Nixon, Walter H. Retired. 98 Zabala Rd., Salinas, Calif. (Vegetable breeding.)

Nolan, Kenneth G. 94 Gardner St., Noroton Heights, Conn.

Nolla, J. A. B. Plant Pathologist, Box 877, Mayagüez, Puerto Rico. (Virology; sugar-cane diseases; disease resistance.)

Nordlinger, Ernest W. Assistant Editor, Food Processing, 111 E. Delaware Pl., Chicago 11, Ill. (Food crop diseases; fungicides.)

Norris, Dale M. Potato Investigation Laboratory, University of Florida, Hastings, Fla.

Norton, Don C. Associate Professor of Plant Pathology, Department of Plant Physiology and Pathology, Agricultural and Mechanical College of Texas, College Station, Tex. (Root-rot diseases; nematology; soil microbiology.)

Norton, Robert J. Director, Crop Protection Institute, Durham, N. H. (Fungicides and insecticides, toxicology and mode of action.)

Nour-Eldin, Farid. Plant Pathologist, Plant Pathology Section, Ministry of Agriculture, P. O. Orman, Egypt. (Virology.)

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Nusbaum, Charles J. Professor of Plant Pathology, North Carolina State College, P. O. Box 5397, State College Station, Raleigh, N. C. (Tobacco diseases; nematode diseases.)

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O'Bannon, John H. Nematologist, Cotton Research Center, Box 815C, Route 2, Tempe, Ariz. (Nematode control measures.)

O'Brien, Muriel J. Plant Pathologist, Potato, Onion, and Root Crop Section, Vegetables and Ornamentals Research Branch, Crops Research Division, Plant Industry Station, Beltsville, Md. (Potato and spinach diseases.)

Ogawa, Joseph Minoru. Assistant Plant Pathologist and Lecturer in Plant Pathology, Department of Plant Pathology, University of California, Davis, Calif. (Diseases of deciduous fruit trees and hops.)

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Olson, Conrad J. Plant Pathologist, Yoder Brothers, Inc., Ashtabula, Ohio. (Ornamentals; chrysanthemum and carnation viruses.)

Olson, Edward O. Plant Pathologist, Citrus Rootstock Investigations, Agricultural Research Service, United States Department of Agriculture, Box 144, Weslaco, Tex. (Citrus and avocado diseases.)

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Orlob, Gert. Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Virology; cereal diseases.)

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Ortega Cantero, Benjamin. Ave Hidalgo 536 Pte, Torreon, Coah, Mexico.

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Oshima, Nagayoshi. Research Fellow, Department of Botany and Plant Pathology, Colorado State University, Ft. Collins, Colo. (Physiology of the fungi; virology; forest tree diseases; potato diseases; cereal diseases.)

Ostazeski, Stanley A. Plant Pathologist, United States Department of Agriculture, Department of Plant Pathology, University of Florida, Gainesville, Fla. (Diseases of forage and cover crop legumes.)

Oswald, John W. Professor of Plant Pathology and Plant Pathologist, Agricultural Experiment Station, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Potato diseases; plant viruses; serology.)

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Owens, Robert G. Plant Pathologist, Boyce Thompson Institute for Plant Research Inc., Yonkers, N. Y. (Fungicides; physiology of fungi.)

Paddock, William Carson. Director, Escuela Agricola Panamericana, Apartado 93, Tegucigalpa, Honduras, Central America. (Diseases of basic food plants in tropical America.)

Padgett, William H. Graduate Student, Department of Botany and Plant Pathology, Alabama Polytechnic Institute, Auburn, Ala. (Forest pathology; mycology.)

Pady, Stuart M. Head of Department of Botany and Plant Pathology, Kansas State College, Manhattan, Kans. (Aerobiology; plant disease survey; rust epidemiology.)

Page, O. T. Plant Pathologist, Tropical Research Department, United Fruit Company, La Lima, Honduras. (Fungus physiology; fungicides.)

Palm, Elmer Thurman. Plant Pathologist, Crop Protection Institute, Durham, N. H. (Fungicides and nematocides.)

Palmer, Hugh C. Plant Pathologist, Agricultural Chemicals Laboratory, Hercules Powder Company, Wilmington, Del. (Fungicides.)

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Panasenko, Vasil T. Professor of Plant Pathology, 340-17th Ave., San Francisco 21, Calif. (Fungus diseases of cereal crops and sugar beets.)

Pantidou, Maria E. Assistant Mycologist, Botany and Plant Pathology Laboratory, Science Service, Ottawa, Ontario, Canada. (Fungi asosciated with mycorrhiza, mainly Boletaceae.)

Panzer, James David. Assistant Professor of Plant Pathology, Department of Plant Pathology, South Dakota State College, Brookings, S. Dak. (Virology; epidemiology.)

Papavizas, George C. Mycologist, Vegetables and Ornamentals Research Branch, United States Department of

Agriculture, Plant Industry Station, Beltsville, Md. (Microecology of soil-borne diseases; soil fungi.)

Pappelis, Aristotel J. Plant Physiologist, Agricultural Marketing Service, United States Department of Agriculture, 13601 Cutler Rd., Miami 56, Fla. (Physiology of parasitism.)

Pappo, Shimon. Plant Pathologist, Plant Protection Division, The Ministry of Agriculture, Kugel Blvd. 14, Entrance No. 3, Holon, Israel. (Diseases of fruit crops.)

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Parmeter, John R., Jr. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of California, Berkeley, Calif. (Forest tree diseases; forest fungi.)

Parr, Thaddeus, Supervisor of Field Research, Chemagro Corporation, 437 Fifth Ave., New York 16, N. Y. (Fungicides.)

Parrado, Jorge L. Estacion Exper. Agronomica, Santiago de Las Vegas, Cuba.

Parris, G. Keith. Professor and Department Head, Department of Botany, Mississippi State College, State College, Miss. (Teaching; mycology; breeding for disease resistance; fungicides.)

Parsons, John L. Senior Experimental Aide, Irrigation Experiment Station, Prosser, Wash. (Stone fruit diseases; viruses.)

Partridge, Arthur D. Forest Pathologist, United States Forest Service, University of Missouri, Room 102, Building T-9, Columbia, Mo. (Tree diseases; fungus variations.)

Partyka, Robert E. Assistant Professor, Extension Plant Pathologist, Department of Botany and Plant Pathology, Ohio State University, Columbus 10, Ohio. (Potatoes and vegetable diseases; nematode problems.)

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Patterson, Fred L. Professor of Agronomy, Department of Agronomy, Purdue University, Lafayette, Ind. (Cereal diseases and breeding for disease resistance.)

Patton, Robert F. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Forest pathology; forest tree improvement.)

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Peleg, Jacob. Director, Agricultural Research Station, Rehovot, Israel.

Pelet, Francis. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Tissue culture; insect transmission of diseases; potato viruses.)

Pelletier, E. Neil, Plant Pathologist, Agricultural Chemicals Laboratory, Hercules Powder Company, Wilmington, Del. (Fungicides; tomato diseases; blueberry diseases.)

Pelletier, Réal L. Assistant Professor of Plant Pathology, Department of Plant Pathology, Macdonald College, Ste. Anne de Bellevue, Quebec, Canada. (Physiology of disease, fungicides.)

Peltier, George L. (Emeritus) Emeritus Professor of Bacteriology, University of Nebraska; Consultant, Indian Trail, Inc., Wisconsin Rapids, Wis. (Cranberry diseases.)

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Peterson, Joseph L. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Vegetable diseases.)

Peterson, L. C. Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Potato diseases.)

Peterson, Paul Donald. Technical Director, Agricultural Sales, Stauffer Chemical Company, Chauncey, N. Y. (Fungicides; insecticides.)

Peturson, B. Dominion Laboratory of Plant Pathology, Winnipeg, Manitoba, Canada.

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Pierce, Walter H. Director, Asgrow Research Center, P. O. Box 793, Twin Falls, Idaho. (Pea and bean breeding and pathology.)

Pieringer, Arthur P. Assistant Horticulturist, Citrus Experiment Station, University of Florida, Lake Alfred, Fla. (Citrus diseases.)

Piersma, Sidney F. Plant Pathologist, California Packing Corporation, Rochelle, Ill. (Vegetable diseases; disease resistance.)

Pierson, Charles F. Plant Pathologist, Agricultural Marketing Service, United States Department of Agriculture, Box 99, P. O. Annex 103, Wenatchee, Wash. (Storage and transit diseases; disease resistance; physiology of parasitism.)

Pinckard, J. A. Plant Pathologist, Shell Chemical Corporation, 119 South Claiborne Ave., New Orleans 12, La. (Soil-borne plant pathogens; chemical control of plant pathogens.)

Pine, Thomas S. Plant Pathologist, Citrus Experiment Station, Agricultural Research Service, United States Department of Agriculture, University of California, Riverside, Calif. (Stone fruit virus diseases.)

Pirone, Pascal P. Plant Pathologist, New York Botanical Garden, New York 58, N. Y. (Diseases of trees, shrubs, and greenhouse plants; air pollution effects on plants.)

Pirone, Thomas P. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Virus diseases; vegetable diseases.)

Pizarro, Antonio C. Plant Pest Control Technologist, Guinobatan Experiment Station, Banao, Guinobatan, Albay, Philippines. (Virology.)

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Poland, George L. Chief Chemist, United Fruit Company Laboratories, Pier 3, North River, New York 6, N. Y. (Tropical fruits.)

Pollard, E. C. Sloane Physics Laboratory, Yale University, New Haven, Conn.

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Pool, Raymond J. (Emeritus) Professor of Botany and Chairman of Department, Emeritus, University of Nebraska, 2845 South 27th St., Lincoln 2, Nebr. (Pathology; ecology; mycology; taxonomy.)

Poole, D. Donald. Director of Agricultural Research, Hunt Foods and Industries, Inc., 131 B St., Hayward, Calif. (Tomato breeding and pathology.)

Poole, Robert Franklin. President, Clemson College, Clemson, S. C. (All phytopathological and mycological matters.)

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Porter, Charles L. Professor of Plant Sciences, Department of Biological Sciences, Purdue University, West Lafayette, Ind. (Industrial mycology; biological control of soil-borne pathogens.)

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Povah, Alfred H. (Life) Science Teacher, Edwin Denby High School, 628 Gladstone Ave., Detroit 2, Mich. (Forest pathology; mycology.)

Powell, Dwight. Professor of Plant Pathology, Department of Plant Pathology, University of Illinois, Urbana, Ill. (Fungicides; fruit diseases.)

Powelson, Robert L. Junior Plant Pathologist, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Small fruit diseases.)

Powers, Harry R., Jr. Plant Pathologist, Crops Research Division, Plant Industry Station, Beltsville, Md. (Cereal diseases.)

Powers, William J. Instructor Adult Education, Atlantic County Vocational Schools, Minotola, N. J. (Instruction of farmers in production of vegetables, fruit, and flowers.)

Pratt, Michael J. Botany Department, Oregon State College, Corvallis, Ore.

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Price, W. C. Virologist, Citrus Experiment Station, Lake Alfred, Fla. (Virology.)

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Qasem, Subhi Abdel-F. Research Assistant, Plant Pathology Department, University of Minnesota, St. Paul 1, Minn. (Grain storage in relation to fungi; fruit and vegetable diseases.)

Qazi, Abdul Quddus. Department of Plant Pathology, University of Wisconsin, Madison 6, Wis.

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Raabe, Robert D. Assistant Professor and Assistant Plant Pathologist, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Diseases of ornamentals; Dematophora root rots; Armillaria.)

Rabie, C. J. Department of Plant Pathology, University of Wisconsin, Madison 6, Wis.

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Raleigh, Walter P. Retired, Clyde, Kans. (Potato diseases.)

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Rands, Robert D. (Emeritus) Retired Head Agriculturist, Rubber Investigations, United States Department of Agriculture; Rubber Plantation Consultant and Citrus Grower, Lake of the Hills, Lake Wales, Fla. (Crude rubber production; citrus diseases.)

Raniere, Lawrence C. Graduate Research Assistant, Department of Plant Pathology, University of Delaware, Newark, Del. (General.)

Rankin, Harvey W. Professor of Plant Pathology and Head of Department, Georgia Coastal Plain Experiment Station, Tifton, Ga. (Seed-borne diseases; vegetable pathology.)

Rankin, W. H. (Life) Bureau of Plant Industry, New York State Department of Agriculture and Markets, Albany, N. Y.

Ranney, Carleton D. Plant Pathologist, Cotton Disease Investigations, Agricultural Research Service, United States Department of Agriculture, Department of Plant Physiology and Pathology, Agricultural and Mechanical College of Texas, College Station, Tex. (Cotton diseases; fungicides; physiology of resistance.)

Raski, Dewey J. Associate Nematologist, Department of Plant Nematology, University of California, Davis, Calif. (Nematodes of grapes and sugar beets; systematics of nematodes.)

Rawlins, Thomas E. (Life) Professor of Plant Pathology and Plant Pathologist in the Experiment Station, Plant Pathology Department, University of California, Berkeley 4, Calif. (Virology; histochemistry.)

Ray, W. Winfield. Chairman and Professor of Botany, Department of Botany, University of Nebraska, Lincoln, Nebr. (Morphology and taxonomy of fungi.)

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Reinking, Otto A. Special Consultant, United Fruit Company, 5213 Worthington Drive, Washington 16, D. C. (Soil Fusaria; soil conditions and root diseases; banana and abaca diseases.)

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Richardson, Lloyd T. Plant Pathologist, Science Service Laboratory, Canada Department of Agriculture, London, Ontario, Canada. (Fungicides.)

Rieman, Gustav H. Professor of Genetics, Department of Genetics, University of Wisconsin, Madison 6, Wis. (Veg-

etable diseases; disease resistance.)

Riffle, Jerry William. Graduate Student in Plant Pathology, Botany and Plant Pathology Department, Michigan State University, East Lansing, Mich. (Forest tree diseases; shade tree diseases.)

Riggs, Robert Dale. Graduate Student, Plant Pathology Department, North Carolina State College, Raleigh, N. C. (Nematology.)

Riker, A. J. (Life) Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Pathological growth; bacterial diseases; diseases of forest trees; research methods; publication problems.)

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Rosen, Harry R. Professor Emeritus of Plant Pathology, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Breeding roses and small grains for disease resistance.)

Rosenberg, Dan Y. Assistant Plant Pathologist, Bureau of Plant Pathology, California Department of Agriculture, 1220 N St., Sacramento 14, Calif. (New disease detection; general plant pathology.)

Rosenkranz, Eugen. Graduate Student, Research Assistant, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Virology, virus diseases of legumes.)

Ross, A. Frank. Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Virus diseases.)

Ross, Eldon Wayne. Graduate Student, Department of Plant Pathology, Bacteriology, and Entomology, West Virginia University, Morgantown, W. Va. (Plant pathology, forest trees.)

Ross, John P. Assistant Professor of Research and Pathologist, United States Department of Agriculture, Department of Plant Pathology, North Carolina State College, P. O. Box 5397, Raleigh, N. C. (Soybean diseases; physiology of parasitism; ornamental diseases.)

Roth, Elmer R. Plant Pathologist, Southeastern Forest Experiment Station, United States Forest Service, Box 2570, Asheville, N. C. (Tree diseases; general.)

Roth, Herbert. Entomologist, Research, Truck Crop and Garden Insects Section, Entomology Research Division, Agricultural Research Service, United States Department of Agriculture, 209 River Street, Hoboken, N. J. (Treatments for plant quarantine.)

Roth, Lewis F. Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, 303 Cordley Hall, Oregon State College, Corvallis, Ore. (Forest diseases.)

Rowell, John B. Plant Physiologist, Crops Research Division, United States Department of Agriculture; Associate Professor of Plant Pathology, Department of Plant Pathology and Botany, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Chemotherapy; physiology of plant disease; fungus physiology.)

Ruehle, Geo. D. Vice-Director in Charge, University of Florida Sub-Tropical Experiment Station, Homestead, Fla. (Diseases of citrus, avocado, mango, and potato.)

Runnels, Harmon A. Assistant Professor, Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Shade tree, ornamental plant, florists' crop, and turf diseases; fungicides.)

Rupert, Joseph A. Director, Chilean Agricultural Program, The Rockefeller Foundation, Casilla 13045, Santiago, Chile, South America. (Cereal breeding and pathology.)

Rusden, Philip L. Plant Pathologist, Bartlett Tree Research Laboratories, 67 Ridgeway Station, Stamford, Conn. (Diseases of shade and ornamental trees and shrubs.)

Russell, R. C. Plant Pathologist, Canada Agriculture Research Laboratory, University of Saskatchewan, Saskatoon, Saskatchewan, Canada. (Cereal diseases, smuts and other seed-borne diseases.)

Ryker, Truman C. Technical Advisor, Grasselli Chemicals Department, E. I. duPont de Nemours & Company, Wilmington 98, Del. (Seed-treatment fungicides.)

Sackett, Robert G. Pathology Student, Agronomy Department, University of Wyoming, Laramie, Wyo. (Vegetable and fruit diseases.)

Sackston, W. E. Plant Pathologist, Plant Pathology Section, Science Service, Canada Department of Agriculture Research Laboratory, University of Manitoba, Box 322, Winnipeg, Mantioba, Canada. (Diseases of oil-seed crops.)

Sakimura, K. Associate Entomologist, Pineapple Research Institute, P. O. Box 3166, Honolulu 2, Hawaii. (Thrips transmission of plant diseases.)

Salisbury, Philip J. Research Officer, Forest Biology Division, Canada Department of Agriculture, 409 Federal Building, Victoria, B. C., Canada. (Root diseases; forest nursery diseases; seed microflora.)

Sander, Evamarie. Graduate Student, Teaching Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Virology.)

Sanford, G. B. (Emeritus) Retired, Plant Pathology Laboratory, Science Service, Canada Agriculture, University of Alberta, Box 913, Edmonton, Alberta, Canada. (General.)

San Juan, Mario O. Instructor in Plant Pathology, College of Agriculture, University of the Philippines, Laguna, Philippines. (Virology.)

Santacroce, Nunzio G. Supervisory Plant Quarantine Inspector in Charge, Plant Quarantine Division, 211 Vierkant, Lisse, The Netherlands. (Plant sciences.)

Santiago, Joaquim Carvalho. Plant Pathologist, Estação de Melhoramento de Plantas, Elvas, Portugal. (Cereal diseases.)

Santilli, Vincent. Assistant Professor of Botany and Research Associate in Botany, Henry Shaw School of Botany, Washington University, St. Louis 5, Mo. (Virology; physiology of parasitism.)

Sasser, J. N. Associate Professor, Plant Pathology, Nematode Diseases, North Carolina State College, Box 5397, Raleigh, N. C. (Phytonematology.)

Saunders, Ward B. Agricultural Commissioner, Court House, San Benito County, Hollister, Calif. (Fruit and vegetable diseases.)

Savage, Earl J. 22 Fairview St., Uniontown, Pa.

Sayed, Mustafa Q. Graduate Student, Room 304, Buckhout Laboratry, University Park, Pa. (Vegetable dis-diseases; storage diseases.)

Sayre, Richard M. Graduate Assistant, Department of Plant Pathology, College of Agriculture, Lincoln, Nebr. (Nematology.)

Schaal, Lawrence A. Senior Pathologist, Agricultural Research Service, United States Department of Agriculture, Department of Botany and Plant Pathology, Colorado State University, Fort Collins, Colo. (Potato disease investigations.)

Schafer, John F. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Disease resistance; cereal diseases; cereal breeding.)

Schafer, Lewis A. Professor and Head of Biology Department, Nebraska Wesleyan University, Lincoln 4, Nebr. (Teaching; cereal and vegetable diseases.)

Schaller, Charles W. Associate Professor of Agronomy, Department of Agronomy, College of Agriculture, University of California, Davis, Calif. (Cereal diseases.)

Scharen, Albert L. Graduate Student, Department of Plant Pathology, University of Nebraska, Lincoln 3, Nebr. (Bacterial diseases; root diseases.)

Scheffer, Robert P. Assistant Professor, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Physiology of parasitism; ornamentals diseases.)

Schein, Richard D. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, The Pennsylvania State University, University Park, Pa. (Cereal diseases; epidemiology.)

Schenck, Norman C. Assistant Plant Pathologist, Watermelon and Grape Investigations Laboratory, University of Florida, P. O. Box 321, Leesburg, Fla. (Vegetable crop diseases; fungicides.)

Schieber, Eugenio. Graduate Student, Plant Pathology Department, University of Wisconsin, Madison 6, Wis. (Cereal crops diseases.)

Schindler, A. F. Associate Nematologist, Nematology Section, Crops Research Division, United States Department of Agriculture, Beltsville, Md. (Nematology; plant pathology, parasitology.)

Schlegel, David E. Assistant Professor of Plant Pathology and Assistant Plant Pathologist in the Experiment Station, Division of Plant Pathology, University of California, Berkeley 4, Calif. (Virology; physiology of disease.)

Schlocker, Archie. Associate Plant Pathologist, Bureau of Plant Pathology, California Department of Agriculture, 1220 N Street, Sacramento 14, Calif. (General pathology; stone fruit virus diseases.)

Schmitt, Chris G. Supervisory Plant Pathologist, Fort Detrick, Frederick, Md. (General plant pathology.)

Schmitthenner, August F. Assistant Professor, Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Diseases of forage legumes and soybeans; soil fungi; root rots; physiology of parasitism.)

Schmitz, Henry. (Life) President, University of Washington, Seattle 5, Wash.

Schnathorst, W. C. Plant Pathologist, Agricultural Research Service, United States Department of Agriculture, Department of Plant Pathology, University of California, Davis, Calif. (Nature of resistance.)

Schneider, Charles L. Plant Pathologist, Sugar Crops, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Sugar beet diseases.)

Schneider, Henry. Associate Plant Pathologist, Department of Plant Pathology, University of California, Riverside, Calif. (Pathological plant anatomy; citrus diseases.)

Schneider, Irving R. Plant Pathologist, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Virology; physiology of plant diseases; chemotherapy.)

Schoeneweiss, Donald F. Research Assistant, Ohio Agricultural Experiment Station, Wooster, Ohio. (Plant pathology.)

Schroeder, Harry William. Plant Pathologist, Quality Maintenance & Improvement Section, Biological Sciences Branch, Marketing Research Division, Agricultural Marketing Service, United States Department of Agriculture, Agricultural Engineering Building, Texas Agricultural and Mechanical College, College Station, Tex. (Cereal diseases; storage diseases.)

Schroeder, Wilbur T. Professor of Plant Pathology, Department of Plant Pathology, New York State Agricultural Experiment Station, Cornell University, Geneva, N. Y. (Disease resistance; vegetable diseases; fungicides; virology.)

Schroth, Milton N. Plant Pathology Department, University of California, Berkeley 4, Calif.

Schuldt, Paul H. Project Leader, Diamond Alkali Company Fellowship; Plant Pathologist, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Fungicides; nematocides; chemical configuration as it relates to biological activity.)

Schuster, M. L. Associate Plant Pathologist, Plant Pathology Department, University of Nebraska, Lincoln 3, Nebr. (Nematode diseases and soil-borne diseases of sugar beets; bacterial diseases of field beans; safflower rust.)

Schwegmann, Jack C. Senior Plant Pathologist, Chalmette Works, Kaiser Aluminum and Chemical Corporation, P. O. Box 1600, Chalmette, La. (Air pollution; diseases of vegetable crops; diseases of ornamental plants.)

Schwinghamer, Erwin A. Plant Pathologist, Biology Department, Brookhaven National Laboratory, Upton,

Long Island, N. Y. (Radiation studies of plant pathogens.)

Scott, Howard A. Assistant Specialist, Department of Plant Pathology, University of California, Berkeley, Calif. (Virology; physiology.)

Seaman, W. L. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Vegetable diseases.)

Segall, R. H. Plant Pathologist, United States Operations Mission to Costa Rica, International Cooperation Administration, USOM, c/o American Embassy, San Jose, Costa Rica. (Diseases of vegetables and tropical plants.)

Sehgal, Om P. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Virology; fungicides.)

Seifter, Eli. Apt. 1-A, 46 Schenck Ave., Great Neck, N. Y.

Self, Raymond Lester. Plant Pathologist, Ornamental Horticulture Field Station, Alabama Polytechnic Institute, P. O. Box 2217, Spring Hill Station, Mobile, Ala. (Diseases of ornamentals.)

Seliskar, Carl E. Chief, Division of Forest Disease Research, Central States Forest Experiment Station, United States Forest Service, 111 Old Federal Building, Columbus 15, Ohio. (Forest pathology.)

Sembi, Lars. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Semeniuk, George. Professor of Plant Pathology, Department of Plant Pathology, South Dakota State College, Brookings, S. Dak. (Legume diseases; root rots; mold physiology.)

Senn, Pettus Holmes. Head Professor of Agronomy, University of Florida, Gainesville, Fla. (Field crops; genetics; plant breeding.)

Sequeira Luis. Pathologist, Coto Research Station United Fruit Company, Golfito, Costa Rica. (Root diseases; soil microbiology.)

Seven, Raymond P. Director of Research, Research Department, Panogen, Inc., Route 14 South, Woodstock, Ill. (Seed treatment; fungicides; grain fumigation; soil fungicides; nematocides.)

Shafer, William B. Assistant Manager, Sales Development Department, Agricultural Chemical Division, Stauffer Chemical Company, 380 Madison Ave., New York, N. Y. (Fungicides; soil fumigants.)

Shah, Muhammad. Graduate Student, Agronomy Department, University of Wyoming, Laramie, Wyo. (General plant pathology.)

Shalla, Thomas A. Research Assistant, Department of Plant Pathology, University of California, Davis, Calif. (Virology.)

Shands, H. L. Professor of Agronomy, Agronomy Building, University of Wisconsin, Madison, Wis. (Small grains and their diseases.)

Shands, R. G. Agronomist, Agronomy Building, University of Wisconsin, Madison 6, Wis. (Barley and wheat breeding; inheritance and linkage of disease resistance.)

Sharp, Eugene L. Assistant Plant Pathologist, Department of Botany and Bacteriology, Montana State College, Bozeman, Mont. (Cereal diseases; fungus physiology.)

Shaw, Charles Gardner. Professor and Plant Pathologist, Department of Plant Pathology, State College of Washington, Pullman, Wash. (Mycology, forest pathology; Peronosporales.)

Shaw, John G. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Virology.)

Shay, J. Ralph. Head, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Fruit pathology; disease resistance; variations in plant pathogens.)

Shea, Keith R. Forest Pathologist, Forestry Research Center, Weyerhauser Timber Company, P. O. Box 420, Centralia, Wash. (Forest tree diseases; dwarf mistletoes; seed molds; root rots; disease resistance.)

Shear, E. V. (Life) Plant Pathologist, Self Employed, Ravena, Albany County, N. Y. (Fruit diseases; diseases of trees and ornamentals; chemical vegetation control.)

Shearer, James T. Research Assistant, Jackson and Perkins Company, Newark, N. Y. (Pathology, nematology, and entomology as related to the nursery business.)

Shepherd, Robert J. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Virology.)

Sher, S. A. Assistant Nematologist, Department of Plant Nematology, Citrus Experiment Station, University of California, Riverside, Calif. (Nematode diseases of plants; nematode taxonomy.)

Sherbakoff, C. D. (Emeritus) Plant Pathologist Emeritus, Agricultural Experiment Station, University of Tennessee, Knoxville 17, Tenn. (The genus Fusarium and related Ascomycetes.)

Sherf, Arden Frederick. Associate Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Vegetable diseases; fungicides; nematodes.)

Sherwood, Robert T. Plant Pathologist, United States Department of Agriculture, Department of Plant Pathology, North Carolina State College, Raleigh, N. C. (Forage crop diseases; root rots.)

Shields, Ivan J. Box 751, Phoenix, Ariz.

Showalter, Joseph W. Inactive Status in Phytopathology, Port Republic, Md.

Shurtleff, Malcolm C., Jr. Extension Plant Pathologist, Botany Hall, Iowa State College, Ames, Iowa. (Fungicides; communications.)

Sideris, Christos P. (Life) 2533 Malama Pl., Honolulu 14, Hawaii.

Siefert, Wayne B. Assistant County Agricultural Agent, 1260 West Blvd., Pontiac, Mich. (Diseases of ornamental plants.)

Siegel, Albert. Assistant Research Botanist, Botany Department, University of California, Los Angeles, Calif. (Virology.)

Siegel, Malcolm R. Plant Pathology Department, University of Delaware, Newark, Del.

Sieveking, Earl G. Manager, Hybrid Seed Corn Division, Funk Brothers Seed Company, Bloomington, Ill. (Hybrid seed corn production; corn breeding; soil fertility.)

Silber, Gustave. Tobacco and Special Crops Section, Plant Industry Station, United States Department of Agriculture, Beltsville, Md.

Silberschmidt, Karl M. Chefe da Secção de Fisiologia Vegetal, Instituto Biológico, Caixa Postal 7119, São Paulo, Brasil. (Virus; potato, vegetables, and ornamentals; immunity.)

Sill, Webster H., Jr. Associate Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Kansas State College, Manhattan, Kans. (Virology; cereal viruses; vegetable diseases.)

Siller, Luis R. Inter-American Institute of Agricultural Sciences, Turrialba, Costa Rica, Central America.

Silverborg, Savel B. Research Associate in Forest Pathology, State University College of Forestry, Syracuse University, Syracuse 10, N. Y. (Plantation tree diseases; hardwood cull; decay in forest products.)

Silverman, William Bernard. Research and Teaching Assistant, Graduate Student, Department of Plant Pathology and Botany, University of Minnesota, St. Paul 1, Minn. (Cereal diseases, especially the rusts.)

Simard, Jacques. Plant Pathologist, Macdonald College, Ste. Anne de Bellevue, Box 147, Quebec, Canada. (Vegetable diseases; fungicides.)

Simmonds, Prye M. Head, Plant Pathology Section, Canada Agriculture Research Laboratory, University Sub Post Office, Saskatoon, Saskatchewan, Canada. (Diseases of cereal crops.)

Simons, John N. Assistant Virologist, Everglades Experiment Station, University of Florida, Belle Glade, Fla. (Insect vectors of virus diseases; virology.)

Simons, Marr D. Plant Pathologist and Associate Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Cereal diseases.)

Simpson, Geddes W. Professor of Entomology, 306 Deering Hall, University of Maine, Orono, Maine. (Insect transmission of potato virus diseases.)

Sims, Asa C., Jr. Assistant Professor of Botany and Plant Pathology, Southern University, Box 9707, Baton Rouge, La. (Fungus physiology.)

Sinclair, James Burton. Assistant Professor of Plant Pathology, Department of Plant Pathology, Louisiana State University, Baton Rouge, La. (Virology; cotton, tomato, and citrus diseases.)

Sinden, James W. Hauser Champignonkulturen Ag., Gossau, Zürich, Switzerland. (Mushroom cultivation, research, and disease control.)

Singh, Ganga Prasad. Assistant Plant Pathologist to Government of Bihar, Agricultural Research Institute, Sabour, Bihar, India. (Virology.)

Singh, Giri Raj. Graduate Student, Department of Plant Pathology, Louisiana State University, Baton Rouge 3, La. (Disease of sugar plants.)

Sisler, Hugh D. Assistant Professor of Plant Pathology, Botany Department, University of Maryland, College Park, Md. (Fungicides; physiology of fungi; viruses.)

Sisson, Max A. District Supervisor, Plant Pest Control Branch, Agricultural Research Service, United States Department of Agriculture, Box 145, Palisade, Colo. Virus diseases of stone fruits; inspection and control work.)

Sitterly, Wayne R. Assistant Plant Pathologist, Clemson College Truck Crop Experiment Station, P. O. Box 3158, Charleston, S. C. (Fungicides; disease resistance; fungus physiology; virology.)

Skaptason, J. B. Manager, Agricultural Products Development, Spencer Chemical Company, Dwight Building, Kansas City, Mo.

Skaptason, Johann S. Field Research, Chemagro Corporation, 3100 West Lake St., Minneapolis 16, Minn. (Seed and soil fungicides.)

Skiles, Robert L. Associate Plant Pathologist, The Rockefeller Foundation, Apartado aereo 58-13, Bogota, Colombia, South America. (Diseases of wheat, barley, corn, potatoes, beans, rice, and forage crops.)

Skolko, A. J. Assistant Chief, Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario, Canada.

Skoog, Harold A. Research Agronomist, Tobacco Experiment Station, Route No. 5, Greeneville, Tenn. (Tobacco diseases.)

Skoropad, William Peter. Associate Plant Pathologist, Plant Pathology Laboratory, Science Service, Canada Department of Agriculture, University of Alberta, Box 913, Edmonton, Alberta, Canada. (Barley diseases.)

Skotland, Calvin B. Assistant Plant Pathologist, Irrigation Experiment Station, Washington State College, Prosser, Wash. (Diseases of irrigated field, vegetable, and specialty crops.)

Slack, Derald A. Associate Professor of Plant Pathology, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Plant parasitic nematodes; fruit diseases.)

Slade, Martin A. Research Bacteriologist and Plant Pathologist, Florida Division, Food Machinery and Chemical Corporation, Lakeland, Fla. (Fungicides; fruit and vegetable diseases.)

Slagg, C. M. (Patron) Botany and Plant Pathology Department, Kansas State College, Manhattan, Kans. (Plant diseases, especially tobacco and horticultural crops.)

Sleeth, Bailey. Pathologist, Texas Agricultural Experiment Station, Weslaco, Tex. (Citrus diseases and ornamentals.)

Slipp, Albert Wiswell. Assistant Professor, Research, College of Forestry, University of Idaho, Moscow, Idaho. (White pine blister rust; ecology of forest fungi; general forest pathology; forest pathology education.)

Slykhuis, John T. Plant Pathologist, Laboratory of Plant Pathology, Science Service, Carling Ave., Ottawa, Ontario, Canada. (Cereal diseases, virus.)

Small, Cyril G. (Life) County Agricultural Agent, Wayne County Extension Service, Alton, N. Y. (Fruit diseases and insects.)

Smalley, Eugene Byron. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Forest pathology; mycology; garlic diseases.)

Smart, Grover C., Jr. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Nematology.)

Smith, Albert L. Plant Pathologist, United States Department of Agriculture, Agronomy Department, Comer Hall, Alabama Agricultural Experiment Station, Auburn, Ala. (Fusarium wilt; bacterial blight; nematodes; breeding for resistance.)

Smith, Cecil G. Box 81, San Juan de los Morros, Venezuela, South America.

Smith, Floyd F. Head, Truck Crop and Garden Insect Section, Entomology Research Service, Plant Industry Station, Beltsville, Md. (Virology; insect vectors.)

Smith, Frank O. Associate Professor of Plant Pathology, Assistant Dean, Faculty of Agricultural Sciences, American University of Beirut, Beirut, Lebanon. (Diseases of fruits and vegetables; survey of plant diseases of the Middle East.)

Smith, Glenn E. Instructor, Department of Botany and Plant Pathology, The Ohio State University, Columbus 10, Ohio. (Antibiotics and nematology.)

Smith, Harlan Eugene. Extension Plant Pathologist, Agricultural Extension Service, College Station, Tex. (General, all plant diseases.)

Smith, Harry L. Regional Supervisor, Eastern Region, Plant Pest Control Division, Agricultural Research Service, United States Department of Agriculture, P. O. Box 93, Moorestown, N. J. (Control and regulatory work on insect pests, plant diseases, and nematodes.)

Smith, Kenneth M. Director of Virus Laboratory, Virus Research Unit, Agricultural Research Council, Cambridge, England. (Virology.)

Smith, Marion A. Senior Pathologist, Agricultural Marketing Service, United States Department of Agriculture, 5630 Ingleside Ave., Chicago 37, Ill. (Market diseases of fruits and vegetables.)

Smith, Oliver Fuhriman. Research Agronomist, Agricultural Experiment Station, Agricultural Research Service, United States Department of Agriculture, University of Nevada, Reno, Nev. (Alfalfa diseases, and breeding alfalfa for resistance to diseases.)

Smith, Paul G. Association Professor of Vegetable Crops, Department of Vegetable Crops, University of California, Davis, Calif. (Vegetable diseases.)

Smith, Thomas E. Director of Plant Breeding, McNair's Yield-Tested Seed Company, Laurinburg, N. C. (Tobacco diseases; genetic resistance.)

Smith, Wilson L., Jr. Vegetable and Field Crops Division, United States Plant Industry Station, Beltsville, Md.

Smoot, John J. Plant Pathologist, Agricultural Marketing Service, United States Department of Agriculture, 2120 Camden Rd., Orlando, Fla. (Post-harvest diseases; epidemiology.)

Smucker, Silas J. Soil Conservationist, Soil Conservation Service, Rensselaer, Ind. (Soil conservation.)

Snell, Walter H. (Life) Stephen T. Olney Professor of Natural History; Chairman, Department of Botany, Brown University, Providence 12, R. I. (Forest pathology; mycology.)

Snow, Glenn A. Pathologist, Forest Disease Laboratory, Post Office Box 122, Gulfport, Miss. (Forest disease research.)

Snow, Gordon Franklin. Assistant Plant Pathologist, Bureau of Plant Pathology, California State Department of Agriculture, 1220 N St., Sacramento 14, Calif. (Virology.)

Snow, Jean A. Graduate Student, 204 Buckhout Laboratory, University Park, Pa. (Cereal diseases.)

Snyder, H. Donald. Chief, Microbiological Laboratory, Troy Chemical Company, 338 Wilson Ave., Newark 5, N. J. (Fungicides and bactericides, industrial applications primarily.)

Snyder, William C. Professor of Plant Pathology and Plant Pathologist in the Experiment Station, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Vegetable diseases; root diseases; fungi.)

Solheim, Wilhelm Gerhard. Professor of Botany, Department of Botany, University of Wyoming, Laramie, Wyo. (Rocky Mountain fungi; genera of Hyphomycetes.)

Somerville, Austin Merchant, Jr. Agricultural Chemicals Development, Virginia-Carolina Chemical Corporation, 401 E. Main St., Richmond 8, Va. (Phytopathology.)

Sommer, Noel F. Assistant Pomologist, Department of Pomology, University of California, Davis, Calif. (Market diseases of fruit.)

Sosa, Oscar N. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Sova, Carl R. Botany and Plant Pathology Department, Ohio State University, Columbus, Ohio.

Sowell, Grover, Jr. 1823 Gresham Rd., S. E., Atlanta, Ga. (Vegetable diseases; fungicides; bacterial diseases.)

Spalding, Donald Hood. Research Assistant, Department of Plant Pathology, Washington State College, Pullman, Wash. (Diseases of field crops and their control; preservation of industrial products.)

Spaulding, Perley. (Charter, Life) 335 Prospect St., New Haven 11, Conn. (Forest tree diseases of the world.)

Spears, Richard K., Jr. Associate Professor of Botany and Forestry, Department of Biology, Centenary College, Shreveport, La. (Forest pathology, morphology, and taxonomy of fungi.)

Spencer, Earl F. Fieldman, Idaho Crop Improvement Association, Inc., Box 188, Idaho Falls, Idaho. (Cereal diseases; diseases of forage crops, virology, potato diseases.)

Spiegelberg, C. H. Plant Pathologist, Pineapple Research Institute, P. O. Box 3166, Honolulu 2, Hawaii. (Diseases of the pineapple plant and fruit.)

Spilker, Oren W. Agricultural Specialist Supervisor, Division of Plant Industry, Ohio Department of Agriculture, Columbus 15, Ohio. (Forest and shade tree diseases; ornamental diseases; regulatory.)

Sprague, Roderick. (Life) Plant Pathologist, Tree Fruit Experiment Station, Route 4, Wenatchee, Wash. (Fungus tree fruit diseases; snow mold of wheat; leaf spots of grasses and grass-like plants.)

Sproston, Thomas, Jr. Professor of Botany, Plant Pathologist, Hills Building, University of Vermont, Burlington, Vt. (Mycology, Sclerotiniaceae; natural disease resistance; polyphenols.)

Spurr, Harvey Wesley, Jr. Graduate Student, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Cereal pathology.)

Srivastava, Daya Nand. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Sugarcane and vegetable diseases; physiology of fungi.)

Staffeldt, E. E. New Mexico College of Agricultural and Mechanical Arts, Box 97, State College, N. Mex.

Stahmann, Mark A. Professor of Biochemistry, Biochemistry Department, University of Wisconsin, Madison 6, Wis. (Biochemistry of plant diseases and disease resistance; proteins and polypeptides.)

Stakman, E. C. (Life) Special Consultant for Agriculture, The Rockefeller Foundation; Professor Emeritus, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Plant pathology and international agricultural improvement.)

Stall, Robert E. Plant Pathologist, Indian River Field Laboratory, P. O. Box 1351, Fort Pierce, Fla. (Diseases of vegetable crops.)

Stambaugh, William J. Assistant Professor of Forest Pathology, Department of Botany and Plant Pathology, The Pennsylvania State University, University Park, Pa. (Tree diseases.)

Standifer, Marie Scott. Biological Aide, Section of Nematology, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Nematology; plant anatomy.)

Stanley, Wendell M. Director of the Virus Laboratory, Professor of Biochemistry, Virus Laboratory, University of California, Berkeley 4, Calif.

Staples, Richard C. Assistant Biochemist, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Cereal diseases; fungal physiology.)

Starkey, Robert Lyman. Professor and Research Specialist in Agricultural Microbiology, Department of Agricultural Microbiology, Agricultural Experiment Station, New Brunswick, N. J. (Agricultural microbiology, including relations of antibiotics to control of plant diseases; microbial physiology.)

Starr, G. H. Agricultural Extension, University of Wyoming, Laramie, Wyo.

Starr, Mortimer P. Associate Professor of Bacteriology and Associate Bacteriologist in the Agricultural Experiment Station, Department of Bacteriology, University of California, Davis, Calif. (Metabolism and taxonomy of phytopathogenic bacteria.)

Starzyk, Marvin J. Graduate Student in Plant Pathology, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Fruit diseases; fungicides.)

Stathis, Panaghiotis D. Graduate Research Assistant, Botany Department, Louisiana State University, Baton Rouge, La. (Fungus diseases of plants and control; virus diseases of plants.)

Steere, Russell L. Assistant Research Biologist, Virus Laboratory, University of California, Berkeley 4, Calif. (Virology.)

Steib, R. J. Associate Plant Pathologist, Botany, Bacteriology, and Plant Pathology Department, Louisana State University, Baton Rouge, La. (Sugar cane diseases.)

Stessel, George J. Assistant Research Professor of Plant Pathology, Department of Plant Pathology-Entomology, University of Rhode Island, Kingston, R. I. (Diseases of ornamental plants and forest trees; nematode diseases of ornamental and forage crops.)

Stevens, Lewis F. Agricultural Chemicals Development, Corona Chemical Division, Pittsburgh Plate Glass Company, New Albany Rd., Moorestown, N. J. (Plant disease control and related fields.)

Stevens, Russell B. Professor of Botany, George Washington University, Washington 6, D. C.

Stevenson, Frederick J. Director of Research, Red Dot Foods, Inc., 1435 E. Washington Ave., Madison 3, Wis. (Genetics; plant breeding; plant pathology.)

Stevenson, John A. Head, Mycology and Plant Disease Reporting Section, Plant Industry Station, Beltsville, Md. (Mycology.)

Stewart, Dewey. Head of Section, Sugar Beet Section, Plant Industry Station, Beltsville, Md. (Sugar beet improvement.)

Stewart, Donald M. Plant Pathologist, Plant Pest Control Division, Agricultural Research Service, United States Department of Agriculture, Room 102, Agricultural Botany Building, University of Minnesota, St. Paul 1, Minn. (Cereal rusts.)

Stewart, R. N. Plant Industry Station, Beltsville, Md.

Stewart, Robert B. Assistant Professor of Plant Pathology, Department of Plant Physiology and Pathology,

Agricultural and Mechanical College of Texas, College Station, Tex. (Field crop diseases.)

Steyaert, René Léopold. Research Mycologist to the Institut National pour l'Étude Agronomique du Congo Belge, 159 Rue des Atrébates, Bruxelles 4, Belgium. (Systematic mycology, principally of tropical fungi and for those connected with plant pathology.)

Stoddard, David L. Research Supervisor, Grasselli Chemicals Department, duPont Experimental Station, Wilmington, Del. (Fungicides; nematocides; insecticides.)

Stoddard, Ernest M. Plant Pathologist, Connecticut Agricultural Experiment Station, New Haven, Conn. (Greenhouse and nursery diseases; chemotherapy.)

Stokes, Granville W. Associate Plant Pathologist, Assistant Professor, Agronomy Department, University of Kentucky, Lexington, Ky. (Tobacco breeding, genetics, and cytogenetics.)

Stoner, Warren N. Assistant Professor of Research Plant Pathology-Entomology, Taft Laboratory, University of Rhode Island, Kingston, R. I. (Virology; insect transmitted plant diseases; economic entomology; ecology.)

Storey, Harold Haydon. (Life) Plant Pathologist, East African Agriculture and Forestry Research Organization, Box 21, Kikuyu, Kenya, East Africa. (Virus diseases; cereal rusts.)

Storm, Leonard W. Student, 132 Hilgard Hall, University of California, Berkeley 4, Calif. (Orchard crop diseases; techniques in plant virology.)

Stouffer, Richard Franklin. Graduate Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Virology.)

Stout, Gilbert Leonidas. Plant Pathologist and Chief, Bureau of Plant Pathology, California Department of Agriculture, Room 345, Department of Agriculture Building, 1220 N St., Sacramento 14, Calif. (Virus diseases of fruit, nut trees, and grapevines; disease surveys; epidemiology; prevention of the introduction of diseases into new areas; certification; administrative and regulatory plant pathology.)

Stover, R. H. Plant Pathologist, Tropical Research Department, United Fruit Company, La Lima, Honduras. (Banana diseases; soil-borne pathogens; soil microbiology.)

Stover, W. C. (Life) Department of Botany, Ohio State University, Columbus 10, Ohio.

Strobel, James Walter. Graduate Student, Washington State College, Pullman, Wash. (Ornamental diseases, foliage plants.)

Strong, Forrest C. Associate Professor, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Forest and shade tree diseases.)

Strong, Miriam C. Assistant Professor, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Vegetable diseases, tomatoes in particular.)

Stroube, William H. Associate Agronomist, Department of Agronomy, Agricultural Experiment Station, Lexington 29, Ky. (Diseases and production of forages.)

Struble, F. Ben. Professor of Plant Pathology, Department of Botany and Plant Pathology, Oklahoma State University, Stillwater, Okla. (Diseases of fruits and vegetables; nematodes.)

Stutzman, Roy H. Sales Supervisor, Panogen Company, Ringwood, Ill. (Seed treatment; stored grain fumigation.)

Suhovecky, Albert J. Plant Pathologist, Agricultural Research Laboratory, Monsanto Chemical Company, Lindbergh and Olive Street Rd., St. Louis 24, Mo. (Fungicides; soil-borne diseases.)

Suit, R. F. Plant Pathologist, Citrus Experiment Station, University of Florida, Lake Alfred, Fla. (Citrus diseases.)

Sullivan, D. M. Engineer, 1404 Johnston Dr., Oklahoma City, Okla. (Plant proteins and virus diseases.)

Summers, Eaton M. In Charge Cane Research, United Fruit Sugar Company, Preston, Oriente, Cuba. (Sugarcane agriculture; breeding; virus strains.)

Summers, Thomas E. Plant Pathologist, United States Department of Agriculture, Belle Glade, Fla. (Diseases of stem and leaf fiber crops.)

Sutton, R. Murray D. Associate Plant Pathologist, Botany and Plant Pathology Laboratories, Science Service, Carling Ave., Ottawa, Ontario, Canada. (Bacterial plant pathogens, methods for their detection, e.g., bacteriophage, identification and control.)

Swank, George, Jr. Plant Pathologist, Agricultural Research and Development, Department 719, The Upjohn Company, Kalamazoo, Mich. (Fungicides.)

Swartwout, Harold G. Professor of Horticulture, Department of Horticulture, University of Missouri, Columbia, Mo. (Diseases horticultural crops, chemical control.)

Swift, Marjorie E. Research Mycologist, Research Division, American Cyanamid Company, Pearl River, N. Y. (Antibiotics; plant growth substances.)

Swingle, Roger U. Plant Pathologist, Ornamentals Section, Agricultural Research Service, United States Department of Agriculture, P. O. Box 70, Worthington, Ohio. (Diseases of trees and woody shrubs.)

Syamananda, Riksh. Plant Pathologist, Bangkhen Agricultural Experiment Station, Bangkok, Thailand. (Diseases of field crops; fungus physiology.)

Sylvester, Edward S. Associate Entomologist, University of California, Berkeley, Calif. (Insect vectors of plant viruses.)

Szkolnik, Michael. Associate Professor of Plant Pathology, New York State Agricultural Experiment Station, Cornell University, Geneva, N. Y. (Pathology and control of fruit diseases; fungicides; antibiotics; chemotherapy.)

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Tamaoki, Taiki. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Physiology of plant diseases; tissue culture.)

Tamburo, Salvatore E. Graduate Student, Department of Plant Pathology, West Virginia University, Morgantown, W. Va. (Sub-tropical fruit pathology; mycology; physiology.)

Tammen, James. Assistant Professor of Plant Pathology, Department of Botany and Plant Pathology, The Pennsylvania State University, University Park, Pa. (Diseases of ornamental plants; taxonomy and physiology of the Fusaria.)

Tanner, Carl R. Research Specialist, California Spray-Chemical Corporation, 2300 S.E. Harvester Dr., Portland 22, Ore. (Fungicides.)

Tarjan, Armen C. Associate Nematologist, Citrus Experiment Station, Lake Alfred, Fla. (Plant nematology.)

Tate, H. Douglas. Manager, Research and Development, Agricultural Chemicals, Naugatuck Chemical, Bethany 15, Conn. (Fungicides.)

Taylor, Albert L. Head Nematology Section, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Plant nematology.)

Taylor, Carlton F. Professor of Plant Pathology and Chairman of Section, Plant Pathology Extension, 211 Buckhout Laboratory, The Pennsylvania State University, University Park, Pa. (Fruit diseases; fungicides; ecology of infection.)

Taylor, Donald P. Instructor, Nematology, Department of Plant Pathology, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Plant parasitic nema-

todes; nematode taxonomy, ecology, relation to other diseases, and control.)

Taylor, Gordon S. Agricultural Scientist, Tobacco Laboratory, Connecticut Agricultural Experiment Station, P. O. Box 348, Windsor, Conn. (Tobacco diseases.)

Taylor, Jack. Assistant Plant Pathologist, Georgia Mountain Experiment Station, Blairsville, Ga. (Fungus diseases of fruits and vegetables.)

Templeton, George E. Assistant Professor of Plant Pathology, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Cereal diseases.)

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Theis, Thomas N. Assistant Officer in Charge, Federal Experiment Station, United States Department of Agriculture, Mayaguez, Puerto Rico. (Diseases of cereal crops and bananas.)

Thirumalacher, M. J. Hindustan Antibiotics Private, Ltd., Poona, India.

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Thomas, H. Earl. Plant Pathologist, 145 Hilgard Hall, University of California, Berkeley 4, Calif. (Diseases of deciduous fruit trees.)

Thomas, H. Rex. Assistant Director, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Vegetable diseases.)

Thomas, Harold E. Director and Pathologist, The Strawberry Institute of California, Cochran Rd., Morgan Hill, Calif. (Strawberry diseases; development of new strawberry varieties.)

Thomas, John E. Professor of Botany and Plant Pathology, Department of Botany and Plant Pathology, Oklahoma State University, Stillwater, Okla. (Tree and ornamental diseases; slime molds; lichens.)

Thomas, Samuel O. Research Mycologist, Lederle Laboratories, Inc. Division American Cyanamid Company, Pearl River, N. Y. (Antibiotics.)

Thomas, Walter Dill, Jr. Lead Research Biologist, California Spray-Chemical Corporation, Ortho Way and Lucas Ave., Richmond, Calif. (Fungicides; bactericides; viricides; insecticides; herbicides; nematocides.)

Thomason, Ivan J. Assistant Nematologist, Department of Plant Nematology, Citrus Experiment Station, University of California, Riverside, Calif. (Biology and control of nematodes attacking vegetable and field crops; developing nematode resistant crops plants; taxonomy of root-knot nematodes.)

Thompson, George E. Professor of Plant Pathology, Department of Plant Pathology, University of Georgia, Athens, Ga. (General plant pathology; forest pathology.)

Thornberry, H. H. (Life) Professor of Plant Pathology, Department of Plant Pathology, University of Illinois, Urbana, Ill. Virology; biochemistry of diseases.)

Thorne, Gerald. Professor of Plant Pathology and Zoology, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Nematology.)

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Tinline, Robert D. Plant Pathologist, Canada Agriculture Research Laboratory, Saskatoon, Saskatchewan, Canada. (Cereal diseases; genetics and physiology of fungi.)

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Travis, Robert V. Horticulturist, Crops Research Division, Agricultural Research Service, Plant Industry Station, Beltsville, Md. (Vector relationships of plant viruses; diseases of ornamentals.)

Treshow, Michael. Plant Pathologist, Agricultural Division, Columbia-Geneva Steel Division, United States Steel Corporation, Provo, Utah. (Small fruit and stone fruit diseases.)

Trione, Edward John. Junior Research Plant Pathologist, Department of Agronomy, University of California, Davis, Calif. (Physiological pathology; physiology of host-parasite relations.)

Troutman, Joseph L. Assistant Professor of Plant Pathology and Physiology, Tobacco Research Station, Chatham, Va. (Tobacco diseases.)

Troxel, Allen W. Instructor in Plant Pathology, Department of Botany and Plant Pathology, Ohio State University, Columbus 10, Ohio. (Virology; bacterial diseases; teaching.)

True, Rodney Philip. Professor of Plant Pathology, Brooks Hall, West Virginia University, Morgantown, W. Va. (Vascular wilt diseases; diseases of turpentine pines; sequence of fungi in dying wood.)

Trujillo, Eduardo E. Graduate Student, Plant Pathology Department, University of California, Berkeley 4, Calif. (Fungus diseases.)

Tsao, Peter H. Junior Plant Pathologist, Department of Plant Pathology, University of California, Riverside, Calif. (Root rot of citrus; antibiosis; systemic fungicides; soil microbiology.)

Tuite, John. Professor of Plant Pathology, Department of Botany and Plant Pathology, Purdue University, West Lafayette, Ind. (Forage diseases; virology.)

Tullis, Edgar C. Plant Pathologist, Building T-712, Operations Research Group, Fort Detrick, Frederick, Md. (Crops diseases.)

Turner, Mason Madison. Research Plant Pathologist, P. O. Box 3011, Modesto, Calif. (Soil fungicides; root rots.)

Tveit, Martin T. Laboratory Manager, Biotechnical Laboratory, Swedish Sugar Corporation, Arlov, Sweden. (Antibiotics; antagonism and soil fungi.)

Twomey, James A. Assistant Plant Pathologist, San Luis Valley Branch Experiment Station, Center, Colo. (Potato and vegetable diseases.)

Tyler, Leon J. Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Teaching; diseases of small grains.)

Tyner, L. E. Officer in Charge, Laboratory of Plant Pathology, Canada Science Service, Edmonton, Alberta, Canada. (Diseases of cereals.)

Udeaja, P. E. Graduate Student in Plant Pathology, Macdonald College of McGill University, Ste. Anne de Bellevue, Quebec, Canada. (Field crops and forest pathology.)

Ullstrup, A. J. Professor of Plant Pathology, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Diseases of corn; nature of disease resistance.)

Ulman, Paul T. Assistant Entomologist, Department of Conservation, 311 W. Washington St., Indianapolis 9, Ind. (Regulatory work; diseases of ornamentals.)

Uritani, Ikuzo. Assistant Professor of Biochemistry, Laboratory of Biochemistry, Faculty of Agriculture, Nagoya University, Anjo, Aichi-ken, Japan. (Pathological chemistry of disease by fungi.)

Utter, L. Gordon. Manager Technical Service, Agricultural Chemicals, Research Center, Diamond Alkali Company, Box 348, Painesville, Ohio. (Fungicides; nematocides; pest control.)

Vaartaja, L. Olli. Forest Pathologist, Canada Agriculture Research Laboratory, University Sub Post Office, Saskatoon, Saskatchewan, Canada. (Diseases of tree seedlings; ecological factors.)

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Van Slogteren, E. Laboratorium Voor Bloem, Lisse, The Netherlands.

Vanterpool, Thomas Clifford. Professor of Plant Pathology, Biology Department, University of Saskatchewan, Saskatoon, Saskatchewan, Canada. (Root rots of cereals; diseases of flax and rape, Pythium, Rhizoctonia.)

Van Zinderen Bakker, E. M. Senior Lecturer in Botany, University of the Orange Free State, Bloemfontein, South Africa. (Virology; deficiencies; aerobiology.)

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Varney, Eugene H. Assistant Research Specialist, Department of Plant Pathology, Rutgers University, New Brunswick, N. J. (Small fruit diseases; virology; mycology.)

Vaughan, Edward K. Plant Pathologist, Oregon Experiment Station; Professor of Plant Pathology, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Diseases of vegetables and small fruits.)

Vaughn, John R. Professor of Plant Pathology, Station Plant Pathologist, College of Agriculture, University of Wyoming, Laramie, Wyo. (Bean diseases; fungicides; cereal crop diseases.)

Veenstra, Maurice A. Graduate Student in Plant Pathology, Department of Botany and Plant Pathology, Michigan State University, East Lansing, Mich. (Small fruit diseases.)

Vekemans, Jacques. Plant Pathologist, Directeur de la Station d'Essais de Kaniama, Ineac, B.P. 83, Kaniama, Belgian Congo. (Tobacco and potato pathology.)

Venkata Ram, C. S. Plant Pathologist, United Planters' Association, P. O. Devarshola, The Niligiris, Southern India, India. (Diseases of plantation crops; wilt diseases of plants; fungicides.)

Vermillion, Monroe T. Assistant Professor of Botany, Ohio University, Athens, Ohio. (Diseases of woody plants.)

Vestal, Edgar F. Plant Pathologist, Plant Protection Advisor, ICA-USOM, International Cooperation Administration, Washington 25, D. C. (Fruit and vegetable diseases.)

Vicklund, Richard E. Manager, Sales and Development, Sindar Corporation, 330 West 42nd St., New York, N. Y. (Fungicides; germicides.)

Viennot-Bourgin, Georges. Professeur de Pathologie Végétale, Institut National Agronomique, 16, rue Claude Bernard, Paris 5°, France. (Pathology, especially rusts and smuts.)

Vincent, Monroe M. Secretary-Treasurer, Microbiological Associates, 4846 Bethesda Ave., Bethesda, Md. (Tissue culture; viral antigens; diagnostic antisera.)

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Visor, Frederick. Product Manager, Plant and Specialty Products, Charles Pfizer and Company, Inc., 800 Second Ave., New York, N. Y. (Antibiotics; plant hormones.)

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Wade, Earl K. Extension Plant Pathologist, Department of Plant Pathology, College of Agriculture, University of Wisconsin, Madison 6, Wis. (General; vegetable, grain, forage crop, fruit, flower, and tree diseases.)

Wadley, Bryce N. Plant Pathologist, Crops Research Division, United States Department of Agriculture, Plant Industry Building, Utah State University, Logan, Utah. (Stone fruit virus diseases.)

Wadsworth, Dallas F. Assistant Plant Pathologist, Botany and Plant Pathology Department, Oklahoma State University, Stillwater, Okla. (Diseases of turf grasses and legumes.)

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Wagnon, H. Keith. Associate Plant Pathologist, Bureau of Plant Pathology, California Department of Agriculture, 1220 N St., Sacramento, Calif. (Virus diseases of fruit and nut tree and vine crops.)

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Waite, Benjamin H. Plant Pathologist, Tropical Research Department, United Fruit Company, La Lima, Honduras, Central America. (Banana diseases, tropical plant diseases.)

Waldee, Edward L. Plant Pathology Advisor, United States Operations Mission, Jordan, Department of State, Washington 25, D. C. (Bacterial diseases; diseases of potatoes; diseases of Middle East.)

Walker, Anson R. Professor and Head, Department of Botany, University of Western Ontario, London, Ontario, Canada. (Phytopathology; mycology; soil microbiology.)

Walker, Earnest A. 6903 Carleton Terrace, College Park, Md.

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Walker, Kenneth R. Plant Pathologist, Banana Board Research Department, Kingston Gardens, 10 South Ave., Kingston, Jamaica, British West Indies. (Banana diseases.)

Walkinshaw, Charles H., Jr. Graduate Student, Plant Pathology Department, University of Wisconsin, Madison 6, Wis. (Potato viruses; citrus diseases.)

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Wallace, Henry A. H. Plant Pathologist, Research Laboratory, Canada Department of Agriculture, University of Manitoba, Box 322, Winnipeg, Manitoba, Canada. (Leaf and seed-borne diseases of barley; root rots of cereals.)

Wallace, James M. Plant Pathologist, Department of Plant Pathology, Citrus Experiment Station, University of California, Riverside, Calif. (Virology.)

Wallen, Victor R. Plant Pathologist, Botany and Plant Pathology Laboratory, Science Service Building, Carling Ave., Ottawa, Ontario, Canada. (Antibiotics; fungicides; seed-borne diseases.)

Wallin, Jack R. Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, 102 Botany Hall, Iowa State College, Ames, Iowa. (Crop plant disease forecasting; bacterial diseases; corn diseases; agricultural meteorology.)

Walter, James M. Plant Pathologist, Manatee Station, Gulf Coast Experiment Station, P. O. Box 2125, Bradenton, Fla. (Breeding tomatoes and beans for combined resistances to diseases.)

Walters, Hubert J. Associate Professor of Plant Pathology, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Diseases of soybeans and forage legumes; virology.)

Walton, Gerald Steven. Graduate Student, Department of Plant Pathology, Rutgers University, New Brunswick, N. J. (Fruit and vegetable diseases.)

Wands, Ralph C. Research Chemist, Minnesota Mining and Manufacturing Company, 2301 Hudson Rd., St. Paul 6, Minn. (General.)

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Ward, Calvin H. Graduate Student, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Diseases of forage crops; nematology.)

Warid, Warid A. College of Agriculture, Kehara University, Giza, Egypt.

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Warnock, Stephen J. Plant Pathologist and Geneticist, Research Laboratory, Campbell Soup Company, P. O. Box 356, Davis, Calif. (Breeding for disease resistance; vegetable crop diseases.)

Wasuwat, Slearm L. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Vegetable crops diseases.)

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Watson, Alice J. Mycologist, Mycology and Plant Disease Reporting Service, Plant Quarantine Division, Agricultural Research Service, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Plant quarantines; mycology; taxonomy of Fungi Imperfecti; bacteriology.)

Watson, I. A. Associate Professor of Genetics and Plant Breeding, Faculty of Agriculture, The University of Sydney, Sydney, N.S.W., Australia. (Cereal diseases and breeding for resistance to them.)

Watson, Roscoe D. Associate Plant Pathologist and Associate Professor of Plant Pathology, Department of Plant Pathology, University of Idaho, Moscow, Idaho. (Root rots; virology.)

Watson, William M. Agriculturist, Crops Regulatory Programs, Plant Pest Control Division, Agricultural Research Service, United States Department of Agriculture, Box 1257, Roanoke, Va. (Entomology; pathology.)

Weast, Lucian D. Plant Quarantine Inspector, Room 844, Federal Building, 641 Washington St., New York 14, N. Y. (General pathology.)

Weathers, L. G. Assistant Plant Pathologist, Department of Plant Pathology, University of California, Riverside, Calif. (Citrus diseases; virology.)

Weaver, L. O. Professor of Plant Pathology, State Plant Pathologist, Department of Botany, University of Maryland, College Park, Md. (Fruit diseases; extension and regulatory plant pathology.)

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Weber, George F. (Life) Professor of Plant Pathology, 406 Rolfs Hall, University of Florida, Gainesville, Fla. (Plant pathology; mycology; teaching.)

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pany, Riverton, N. J. (Vegetable diseases; fungicides; breeding for disease resistance.)

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Weinhold, Albert R. Graduate Student, Department of Plant Pathology, University of California, Davis, Calif. (Fungus diseases; fungus physiology.)

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Weintraub, Robert L. Biochemist, United States Biological Warfare Laboratory, Fort Detrick, Frederick, Md. (Biochemistry of pathogenic fungi.)

Weiss, Freeman A. Curator, American Type Culture Collection, 2112 M St., N.W., Washington 7, D. C. (Preservation of cultures of microorganisms and viruses.)

Weitz, Harold. Technical Representative, Agricultural Chemical Sales Division, Shell Chemical Corporation, 119 South Claiborne Ave., New Orleans 12, La. (Soil fumigation; nematology; vegetable pathology.)

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White, Neville H. Associate Professor of Plant Pathology, School of Agriculture, University of Sydney, Sydney, New South Wales, Australia. (Host-pathogen relations, fungi and viruses.)

White, Richard Peregrine. (Life) Executive Vice President, American Association of Nurserymen, Inc., 635 Southern Building, Washington 5, D. C. (Quarantines; federal-state relations; regulatory matters.)

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Whitney, Roy D. Forest Pathologist, Canada Agriculture Research Laboratory, University of Saskatchewan, Saskatoon, Saskatchewan, Canada. (Root diseases.)

Whitney, W. A. (Life) 16 Catherine St., Schenectady, N. Y.

Wiedman, Harold W. Assistant Plant Pathologist, Department of Botany and Entomology, New Mexico College of Agricultural and Mechanical Arts, State College, N. Mex. (Seedling diseases of cotton; effects of crop residues on soil-borne diseases; vegetable diseases.)

Wiehe, Paul Octave. Director, Sugar Research, Institute Réduit, Mauritius. (Agronomy and pathology of the sugar cane plant.)

Wieman, John S. Superintendent, Bureau of Nursery Service, Oregon Department of Agriculture, 372 State Office Building, Portland 1, Ore. (Pests and diseases; inspection nursery stock.)

Wilcox, Louis V., Jr. Graduate Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Ornamental diseases.)

Wilcoxon, Frank. Statistical Consultant, Boyce Thompson Institute for Plant Research, 1086 North Broadway, Yonkers, N. Y. (Applied statistical methods.)

Wilcoxson, Roy D. Instructor, Department of Plant Pathology and Botany, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Forage crop diseases.)

Wilde, Peter. Graduate Student, Research Assistant (Until July, 1958: Department of Plant Pathology, University of Minnesota, St. Paul, Minn.), Institut f. Pflanzenpathologie der Univ. Goettingen, Nikolausberger Weg 5a, Goettingen, Germany. (Physiology and genetics of fungi.)

Wildman, Sam. Botany Department, University of California, Los Angeles 24, Calif.

Wiles, Alfred Barksdale. Plant Pathologist, Department of Plant Pathology and Physiology, Mississippi Agricultural Experiment Station, State College, Miss. (Cotton diseases; disease resistance; seed and soil fungicides.)

Wilhelm, Stephen. Associate Professor of Plant Pathol-

ogy, University of California, Berkeley 4, Calif. (Small fruit pathology; mycology.)

Wilkinson, Robert E. Associate Professor of Plant Pathology, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Vegetable diseases; virology.)

Wilks, Jack M. Associate Plant Pathologist, Plant Pathology Laboratory, Box 1167, Creston, B. C., Canada. (Stone fruit virology.)

Willey, C. R. 1112 State Office Building, Richmond 19, Va.

Williams, A. S. Associate Plant Pathologist, Department of Plant Pathology and Physiology, Virginia Agricultural Experiment Station, Blacksburg, Va. (Diseases of forage crops.)

Williams, Barbara J. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison 6, Wis. (Physiology and genetics of parasitism.)

Williams, Edwin B. Associate Professor of Plant Pathology, Department of Botany and Plant Pathology, Purdue University, Lafayette, Ind. (Fruit pathology; fungicides.)

Williams, Floyd J. Research Assistant, Department of Botany and Plant Pathology, The Ohio State University, Columbus 10, Ohio.

Williams, Harold E. Assistant Plant Pathologist, Bureau of Plant Pathology, State Department of Agriculture, 1220 N St., Sacramento 14, Calif. (Virus diseases of stone fruits.)

Williams, James L., Jr. Botany and Plant Pathology Department, Purdue University, W. Lafayette, Ind.

Williams, Lansing E. Assistant Professor, Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Cereal diseases; soil fungi.)

Williamson, Charles E. Associate Professor of Plant Pathology, Ornamentals Research Laboratory, Cornell University, Farmingdale, N. Y. (Diseases of ornamentals; soil fumigants; nematode diseases.)

Willis, George M. Ohio State University, Columbus, Ohio.

Willison, Robert S. Senior Plant Pathologist, Laboratory of Plant Pathology, Box 506, St. Catharines, Ontario, Canada. (Fruit diseases; virology.)

Wills, Wirt H. Assistant Plant Pathologist, Tobacco Research Station, Chatham, Va. (Tobacco diseases.)

Wilson, Alasdair R. (Life) Plant Pathologist, Scottish Horticultural Research Institute, Invergowrie, Dundee, Scotland, United Kingdom. (Potato storage; fungal and bacterial diseases of potatoes and tomatoes.)

Wilson, Charles L. Department of Plant Pathology, University of Arkansas, Fayetteville, Ark.

Wilson, Edward E. Professor of Plant Pathology and Plant Pathologist, Department of Plant Pathology, University of California, Davis, Calif. (Tree fruit and nut diseases; fungicides.)

Wilson, Elmer L. Plant Quarantine Inspector, Plant Quarantine Division, Agricultural Research Service, United States Department of Agriculture, 3 Anza Dr., Nogales, Ariz. (Plant pathology; biophysics.)

Wilson, Eugene M. Research Assistant, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Plant protection; host-parasite relationships; rust diseases.)

Wilson, J. Dean. Professor of Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Control of foliage and soil-borne diseases, including nematodes, of vegetables.)

Wilson, Jack Belmont. Assistant Professor of Plant Pathology, Department of Botany, University of Maryland, College Park, Md. (Diseases of ornamentals; mycology; diseases of vegetables.)

Wilson, Norton S. 3761 Bandini Ave., Riverside, Calif.

Wilson, V. E. Box 331, Twin Falls, Idaho.

Wilson, William E. Associate Professor of Botany, Department of Botany, Miami University, Oxford, Ohio. Physiology of fungi; teaching.)

Wingard, Samuel Andrew. Plant Pathologist and Head of Department, Department of Plant Pathology and Physiology, Virginia Polytechnic Institute, Blacksburg, Va. (Diseases of beans, tomatoes, fruits, and tobacco; fungicides.)

Winstead, Nash Nicks. Assistant Professor of Plant Pathology, Plant Pathology, North Carolina State College, Box 5397, Raleigh, N. C. (Vegetable diseases; breeding for disease resistance; physiology of diseases.)

Winston, John R. (Emeritus) Senior Horticulturist, Biological Sciences Branch, Agricultural Marketing Service, United States Department of Agriculture, 2120 Camden Rd., Orlando, Fla. (Post-harvest fruit pathology.)

Winter, Floyd L. Vice President, Breeding and Research Department, Associated Seed Growers, Inc., 205 Church St., New Haven, Conn. (Plant breeding; vegetable crop diseases.)

Winter, Henry Frank. Associate Professor of Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio. (Fruit diseases; fungicides.)

Wismer, Chester A. Senior Pathologist, Experiment Station, Hawaiian Sugar Planters' Association, 1527 Kecaumoku St., Honolulu, Hawaii. (Sugar cane pathology.)

Witcher, Wesley. Graduate Student, Plant Pathology Department, North Carolina State College, Raleigh, N. C. (Fruit diseases, blueberries.)

Wolf, Frederick A. (Charter, Life) Professor Emeritus, Duke University, Durham, N. C. (Tobacco diseases; fungi.)

Wolf, Frederick T. Professor of Botany, Department of Biology, Vanderbilt University, Nashville, Tenn. (Mycology; physiology of fungi.)

Wollman, Erwin S. H. Senior Technician, Department of Plant Pathology, Citrus Experiment Station, Riverside, Calif. (Virus, citrus.)

Woo, Ji-Hung. Food Technologist, Agriculture Research Department, California Packing Corporation, 850 Thornton St., San Leandro, Calif. (Crown gall disease of peaches; antibiotics.)

Wood, Jessie I. Plant Disease Survey, Plant Industry Station, Beltsville, Md.

Wood, Leon S. Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Department of Plant Pathology, South Dakota State College, Brookings, S. Dak. (Cereal diseases; soil-borne diseases.)

Woodbury, E. N. Supervisor, Agricultural Chemicals Laboratory, Research Center, Hercules Powder Company, Wilmington, Del. (Fungicides.)

Woodcock, William P. Graduate Assistant, Department of Plant Pathology, Cornell University, Ithaca, N. Y. (Ornamentals; teaching; extension; fungicides; entomology.)

Woods, Mark W. (Biologist, Laboratory of Cytochemistry, National Cancer Institute, National Institutes of Health, Bethesda 14, Md. (Virology, cancer metabolism and chemotherapy.)

Wooley, Paul H. Extension Entomologist, Assistant Professor of Entomology, Department of Entomology, Cornell University, Ithaca, N. Y. (Fruit diseases; virology; nematology.)

Woolliams, G. E. Plant Pathologist, Plant Pathology Laboratory, Box 8, Summerland, B. C., Canada. (Vegetable diseases.)

Wright, Ernest. (Life) Research Pathologist, Oregon Forest Research Center, P. O. Box 409, Corvallis, Ore. (Forest pathology; soil microbiology; root rots.)

Wright, Theodore R. Plant Pathologist, Orchard Supply Distributors, Box 1667, Wenatchee, Wash. (Fungicides.)

Wu, Lung-chi. Graduate Student, Horticultural Field Laboratory, University of Illinois, Urbana, Ill. (Physiology of fungi.)

Wyllie, Thomas D. Research Fellow, Department of Plant Pathology and Botany, Institute of Agriculture, University of Minnesota, St. Paul 1, Minn. (Soybean diseases and related diseases on legumes.)

Wysong, Noel B. Chief Forester, Forest Preserve District, 536 No. Harlem Ave., River Forest, Ill. (Forestry and tree diseases generally.)

Yamamoto, Masaki. Professor of Plant Pathology, Laboratory of Plant Pathology, Shimane Agricultural College, Matsue, Japan. (Potato late-blight; nature of resistance of plant diseases; diseases of rice plant.)

Yarwood, Cecil Edmund. Professor of Plant Pathology and Plant Pathologist, Department of Plant Pathology, University of California, Berkeley 4, Calif. (Obligate parasitism; diurnal periodicity; acquired immunity; virology.)

Yerkes, William D., Jr. Associate Plant Pathologist, in Charge of Field Bean Breeding and Development, Rockefeller Foundation, Calle Londres 40, Mexico 6, D. F., Mexico. (Legume diseases and breeding; vegetable diseases; mycology; virology.)

Yoder, Donald M. Head, Biological Research, Union Carbide Fellowship, Boyce Thompson Institute for Plant Research, Inc., 1086 North Broadway, Yonkers, N. Y. (Mode of action of fungicides; fungicide evaluation.)

Yost, Henry E. Assistant Regional Superintendent, Agricultural Research Service, United States Department of Agriculture, Box 93, Moorestown, N. J. (Plant pest control survey.)

Young, H. C. Professor of Plant Pathology and Associate Chairman of Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Ohio State University, Wooster, Ohio. (Fruit diseases; fungicides; soil fungi.)

Young, H. C., Jr. Professor of Plant Pathology, Department of Botany and Plant Pathology, Oklahoma State University, Stillwater, Okla. (Diseases of cereal and forage crops.)

Young, Paul A. (Life) Plant Pathologist in Charge, Tomato Disease Laboratory, Texas Agricultural Experiment Station, Jacksonville, Tex. (Breeding new tomato varieties for disease control; tomato genetics; chemical treatment of soil to control pests; watermelon diseases.)

Young, Roy A. Professor, Plant Pathologist, Department of Botany and Plant Pathology, Oregon State College, Corvallis, Ore. (Ornamental diseases; potato diseases; fungicides.)

Young, V. H. Professor Emeritus, Department of Plant Pathology, University of Arkansas, Fayetteville, Ark. (Diseases of cotton; fungal enzymes.)

Young, Virgil H., Jr. Specialist Plant Chemicals, Plant Chemical Section, Product Development Department, Chemical Division, Merck and Company, Inc., Rahway, N. J. (Fungicides and nematocides.)

Young, William J. Plant Pathologist, Michigan Department of Agriculture, Lewis Cass Building, Lansing 13, Mich. (Stone fruit viruses; ornamental and tree diseases.)

Younkin, S. G. Director of Agricultural Research, Campbell Soup Company, Camden 1, N. J. (Vegetable diseases.)

Yount, William L. Plant Pathologist, Pennsylvania Department of Agriculture, Harrisburg, Pa. (Diseases of nursery plants and seed; certification of seed and nursery stock; plant disease surveys.)

Yukawa, Yosio. Assistant Professor of Plant Pathology, Faculty of Agriculture, Yamaguti University, Tyohu, Simonoseki-si, Yamaguti-ken, Japan. (Pathological physiology and anatomy, especially of plant galls.)

Zabel, Robert. Chairman, Department of Botany and Forest Pathology, State University College of Forestry, Syracuse University, Syracuse, N. Y. (Products pathology; forest pathology; fungicides.)

Zaitlin, Milton. Assistant Professor of Horticulture, Department of Horticulture, University of Missouri, Columbia, Mo. (Virology; plant proteins.)

Zak, Bratislav. Forest Pathologist, Athens-Macon Research Center, United States Forest Service, c/o Forestry School, University of Georgia, Athens, Ga. (Forest pathology; root diseases; study of mycorrhizae.)

Zambettakis, Charalambos. Professor of Plant Pathology, University Thessaloniki Greece, Chargé de Recherches, C.N.R.S., 9 rue de l'Eglise, Antony, Seine, France. (Plant diseases in general; fungicides; parasitic Imperfect Fungi.)

Zaumeyer, W. J. Principal Pathologist, United States Department of Agriculture, Plant Industry Station, Beltsville, Md. (Bean and pea diseases; disease resistance; antibiotics.)

Zentmyer, George A. Plant Pathologist, Department of Plant Pathology, University of California, Riverside, Calif. (Avocado diseases; mechanism of action of fungicides; chemotherapy; root rots.)

Ziffer, Jack. Head, Department of Microbiology, Pabst Laboratories, 1037 West McKinley, Milwaukee, Wis. (Antibiotics; fungicides; ornamental diseases; forest pathology; mycology; physiology of fungi.)

Zscheile, Frederick Paul, Jr. Professor of Agronomy and Biochemist in the Experiment Station, Department of Agronomy, University of California, Davis, Calif. (Biochemistry and physiology of disease resistance.)

Zuck, Robert K. Professor and Chairman, Department of Botany, Drew University, Madison, N. J. (Mycology.)

Zuckerman, Bert M. Station Pathologist, Cranberry Experiment Station, University of Massachusetts, East Wareham, Mass. (Small fruit diseases; forest pathology; wood deterioration.)

Zummo, Nat. Graduate Student, Department of Botany, Bacteriology, and Plant Pathology, Louisiana State University, Baton Rouge, La. (Turf diseases.)

SUPPLEMENTARY LIST

(not recorded in Geographical Index or Table 1)

Ashworth, Lee J., Jr. Department of Plant Pathology, University of California, Davis, Calif.

Bein, Selwyn J. Box 692, Miami Springs, Fla.

Bergeson, Glenn B. Botany and Plant Pathology Department, Purdue University, Lafayette, Ind.

Bissonnette, H. L. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Bragonier, Wendell H. Professor and Head, Department of Botany and Plant Pathology, Iowa State College, Ames, Iowa. (Teaching; diseases of woody plants.)

Clarkson, M. R. Agricultural Research Service, United States Department of Agriculture, Washington 25, D. C.

Dirks, Henry T. Petawawa Forest Experiment Station, Chalk River, Ontario, Canada.

Eid, Raymond F. School of Agriculture, University of Delaware, Newark, Del.

Farber, Syril B. 79 Martin Court, Jericho, N. Y.

Gattani, M. L. Agricultural Officer, Plant Pathologist, Food and Agricultural Organization of the United Nations, F.A.O. Mission, Kabul, Afghanistan. (Plant pathology and anti-fungal antibiotics.)

Gomez, Juan C. Escuela de Biologia, Ap. Sab. Grad. 10098, Caracas, Venezuela, South America.

Goss, Robert C. Biology Department, Loyola University, New Orleans 18, La.

Hotson, H. H. 3949 15th Ave., N. E., Seattle, Wash.

Lohr, Arthur D. 720 Greenwood Road, Wilmington, Del.

Lortie, Marcel. Graduate Student, Department of Plant Pathology, University of Wisconsin, Madison, Wis. (Forest pathology.)

McGrath, (Mrs.) Hilde. Mycology and Plant Disease Reporting Section, Plant Industry Station, Beltsville, Md.

Martinez, Albert P. Assistant Plant Pathologist, State Plant Board Laboratory, P. O. Box 3777, University Station, Gainesville, Fla. (Ornamental plant pathology.)

Minteer, Robert J. Department of Plant Pathology, Cornell University, Ithaca, N. Y.

Ohman, John H. Department of Plant Pathology, University of Minnesota, St. Paul 1, Minn.

Pedder, D. Lunevale Products, Ltd., Low Mill, Halton, Lancaster, England, United Kingdom.

Rohde, Richard Allen. Graduate Assistant, Department of Botany, University of Maryland, College Park, Md. (Nematology.)

Van Diepen, J. R. Department of Plant Pathology and Botany, Ohio State University, Columbus, Ohio.

CONSTITUTION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

As Revised to January 1, 1958

Article I—Name and Objectives

The name of this Society shall be The American Phytopathological Society. Its objects are to promote: 1) the increase and diffusion of all aspects of knowledge relating to plant diseases and their control, 2) effective working relations between persons professionally engaged in this field of study, and 3) coordination of various interests and services pertinent thereto.

Article II—Membership

Section 1. The Society shall consist of Charter Members, Annual Members, Student Members, Patrons, Sustaining Associates, and Emeritus Members.

Section 2.

(a) *Charter Members.* The Charter Members are the one hundred thirty persons who accepted the invitation of the Organization Committee of October 25, 1909, to form the Society.

(b) *Annual Members.* Any person interested in the study and control of plant diseases shall be eligible for membership. Applications for membership must be endorsed by at least one member of the Society and forwarded to the Treasurer with such dues as may be prescribed in the Standing Rules. Applicants may be elected at any regular meeting of the Society or by the Council in the interim.

(c) *Student Members.* Any person interested in the study and control of plant diseases who is enrolled as a senior or graduate student in an accredited college or university is eligible for student membership for a period not to exceed five years provided he is not gainfully employed more than two-thirds time. Applications must be endorsed by a member of the Society, and the student and employment status must be certified by the major professor or appropriate department head of the educational institution. Student Members are accorded all the tenure and privileges of Annual Members but must renew the student certification each year when submitting to the treasurer such dues as may be prescribed in the Standing Rules, until they complete their academic training or become employed more than two-thirds time when they automatically become eligible for regular Annual Membership. Applicants may be elected at any regular meeting of the Society or by the Council in the interim.

(d) *Patrons.* Any person making a payment to the Society of such amount as may be prescribed in the Standing Rules shall be designated a Patron and, upon election to membership, shall have all the privileges of an Annual Member.

(e) *Sustaining Associates.* Any firm making an annual contribution to the Society of such amount as prescribed in the Standing Rules shall be designated a Sustaining Associate, and shall receive the official journal without charge.

(f) *Emeritus Members.* Members of twenty years' standing may become Emeritus Members upon application to the Secretary. An Emeritus Member shall retain all rights and privileges of the Society and is exempt from payment of dues, but shall not receive Phytopathology unless he so desires, in which case he shall receive it at cost.

Article III—Officers

Section 1. The officers of the Society shall be a President, a Vice President, a Secretary, and a Treasurer.

Section 2. The duties of the officers shall be those customarily pertaining to these offices.

Article IV—Council

Section 1. The government of the Society and its corporate authority as defined in the Society's Articles of Incorporation shall be vested in a Council which shall consist of the contemporary officers, the retiring President, the President-elect, and the Editor-in-Chief and the Business Manager of the Journal, Phytopathology, a Councilor from each unit of the Society that is organized as a Division, and two Councilors-at-large.

Section 2. The President shall call meetings of the Council at his discretion or upon the written request of three of its members. He, or in his absence the Vice President, shall preside at meetings of the Council. In the absence of both the President and Vice President, the President-elect shall preside at meetings of the Council.

Section 3. The presence of a majority of the Council shall constitute a quorum for the transaction of business.

Section 4. All actions of the Council or officers must be authorized or approved by the Society at the annual meeting except as specified in Article VII, Section 3.

Article V—Selection of Officers and Councilors

Section 1. The President-Elect, Vice President, and Councilors-at-Large shall be elected by ballot. The Secretary shall send a nomination ballot for these offices to all members of the Society in time to allow nominations to be returned not less than two months before the date of the annual meeting. The Council shall designate the nominees for any office when such nominations are wanting, or are tied. The names of the three persons who receive the highest number of nominating votes for each office shall be placed on a final ballot, which shall be sent to each resident member not less than one month before the annual meeting. If the same person should receive sufficient ballots to qualify for nomination to more than one

office, his name shall be placed on the final ballot as a candidate for only the office for which he receives the greater number of ballots, and in the case of an equal number of ballots he shall be entered as a candidate for only the higher office as listed in Article III, Section 1. These ballots shall be returned to the Secretary and shall be canvassed by the Council. A plurality vote shall elect.

Section 2. The Secretary and the Treasurer shall be appointed by the Council, ordinarily for terms of three years, which shall not expire concurrently, and the Council may adjust the term or the date of assuming office to avoid this contingency.

Section 3. The President-elect shall become President and assume the duties of the office upon final adjournment of the annual meeting next succeeding the annual meeting at which he is elected. The Vice President shall assume office upon final adjournment of the annual meeting at which he is elected. The President and Vice President shall not be eligible to immediate reelection to the same offices.

Section 4. The terms of the Councilors-at-large shall be two years beginning with the final adjournment of the annual meeting at which they are elected, or until their successors are elected.

Section 5. Division Councilors shall be elected by their respective Divisions in such manner and for such terms as the Divisions may prescribe.

Section 6. The Council shall fill by appointment any vacancy, except that of a Division Councilor, occurring within the prescribed term, such appointment to continue for the unexpired balance.

Section 7. The President shall appoint Alternates-at-Large for the Councilors-at-Large who are unable to attend the official Council meetings.

Article VI—Society Journal and Editorial Board

Section 1. The official publication of the Society shall be the journal Phytopathology.

Section 2. The Council shall appoint an Editor-in-Chief and a Business Manager of the Journal for terms of three years not expiring concurrently, and also an Editorial Board as prescribed in the Standing Rules.

Section 3. The Council may authorize the Editor-in-Chief and the Business Manager to employ such assistants as may be necessary for the proper conduct of their work.

Article VII—Committees and Conduct of Society Business

Section 1. The Council shall appoint such standing and special committees as may be appropriate for conducting the business of the Society.

Section 2.

(a) *Standing Committees.* Committees whose functions include the general policies and internal relations of the Society, and its relations with other organizations, shall be known as Standing Committees. They shall have a revolving membership and shall submit an annual report to the Council at the Society's annual meeting.

(b) *Special Committees.* Committees whose function is to deal with special subjects of concern to the Society shall be designated Special Committees. Each special committee shall be continued for such period as in the judgment of the Council may be necessary for the accomplishment of its purpose. Interim reports may be requested and a final report shall be made to the Council at the close of this period.

(c) *Temporary Committees.* Committees for the accomplishment of a specific purpose, limited in scope and time, may be appointed by the President to serve during his term and to make such reports to the Council as he may direct.

Section 3. In the interim between regular meetings of the Society the Council may undertake and carry out such actions as it deems advisable or necessary, including the reference of particular questions to the Society members by mail or by notices in the official Journal, subject to approval by the Society at its next meeting.

Article VIII—Funds

The control of funds of the Society received from dues, subscriptions to the Journal, gifts, bequests and endowments shall be vested in the Council to administer through the appropriate fiscal officers and committees. An audit of the receipts and disbursements shall be made annually by a temporary committee, or at the discretion of the Council by a certified public accountant. The reports of the Treasurer and the Business Manager, together with that of the auditing committee, shall be published annually in the official journal.

Article IX—Meetings

A general meeting of the Society shall be held each year, unless prevented by a national emergency, at such place, and time as the Council may direct. Special or local meetings for the presentation of scientific papers or demonstrations of experimental results may be arranged at the discretion of the Council.

Article X—Divisions

Section 1. Branch organizations or units within the Society, known as Divisions, may be established within the geographical area of North America, provided that a formal application setting forth the reasons for such establishment is made to, and approved by, the Society.

Section 2. The organizations and the operations of Divisions shall be governed by the specifications relative to Divisions in the Standing Rules.

Article XI—Affiliated Societies

For the purpose of promoting international collaboration between professional organizations in the field

of plant pathology, the Council of The American Phytopathological Society is authorized to establish affiliated relations with other phytopathological societies in accordance with the provisions pertaining thereto in the Standing Rules.

Article XII—Ratification and Amendment

Section 1. This Constitution shall become effective upon its ratification in accordance with the procedure for amending the original Constitution of the Society. It shall supersede the original Constitution and all amendments thereto.

Section 2. This Constitution may be amended at any annual meeting of the Society provided that any proposed amendment be approved by the Council and be published in the official journal, or otherwise communicated to all resident members, at least one month before the annual meeting, and that it receive the affirmative votes of three-fourths of the members voting at a regularly scheduled business session.

Standing Rules

1. dues, arrears, reinstatement

(a) The dues, including subscription to Phytopathology, for all Annual Members as defined in Article II, Section 2 (b) of the Constitution shall be $10.00 a year, and for Student Members, as defined in Section 2 (c), shall be $5.00 a year. In order to qualify for the rate as a Student Member, applicants must submit with their dues each year a certificate of student status endorsed by their major professor or the head of the department of instruction.

(b) All dues shall be payable by December 31, and those who fail to make payment to the Treasurer by this date shall be considered in arrears and dropped from membership. Members in arrears may be reinstated, without the formality of reelection, by payment of current dues and, in the case of students, certification of student status as specified in Section 2 (c) of Article II of the Constitution.

(c) The contribution of a Patron shall be one thousand dollars, payable in one sum.

(d) The annual contribution of a Sustaining Associate shall be at least one hundred dollars, and a list of Sustaining Associates shall be published in each issue of the journal.

(e) The payments received from Patrons and Sustaining Associates shall be credited to the current funds of the Society. These funds shall be transferable to the account of Phytopathology, or may be used for other purposes authorized by the Council.

2. phytopathology

(a) *Editorial Board.* The editorial policy of the official journal, Phytopathology, shall be vested in an Editorial Board consisting of an Editor-in-Chief and fifteen Editors; the business control shall be vested in a Business Manager and an Advertising (or Assistant Business) Manager.

(b) *Selection of Board Members.* This Board, aside from the Editor-in-Chief and the Business Manager, the manner of whose appointment is prescribed in Article VI, Section 2 of the Constitution, shall be selected from the membership by the Council in consultation with the Editor-in-Chief, and approved by the Society.

(c) *Term of Office.* The term of each member of the Board shall be three years and the appointments, exclusive of the Editor-in-Chief, shall be staggered so that five members are appointed each year. The Council is authorized to make any adjustments in these terms of office necessary to effect non-concurrence.

(d) *Compensation of Advertising Manager.* The Advertising Manager may receive compensation for his services on a commission basis as determined by the Council.

(e) *Subscriptions and Back Numbers.* Subscriptions to Phytopathology shall be $10.00 per year, domestic and foreign. The price of single current numbers, within 60 days of publication, shall be $1.00. The sale and price of back volumes or numbers shall be determined by the Business Manager with the approval of the Council. Requests to supply lost copies of the journal without charge must be made within sixty days from date of issue (January 15 and monthly thereafter).

(f) *Income from advertising.* Income received from the sale of advertising space in Phytopathology shall be devoted to the production of the Journal.

3. program committee

The program for the annual meeting shall be in charge of a temporary committee consisting of the President, Vice President, Secretary, Editor-in-Chief, and such additional members as the President may select. This committee shall have full authority over the scheduling of sessions and demonstrations, and the allocation of papers.

4. expenses of officers at annual meeting

The Secretary, the Treasurer, the President or in his place the Vice President, are authorized to receive reimbursement from the Society for travel expenses in connection with their attendance at the annual meeting. In addition the Editor-in-Chief of Phytopathology is also authorized to receive reimbursement for travel expenses to the annual meeting.

5. auditing committee

An auditing committee shall be appointed by the President each year which shall, prior to the annual meeting, audit the accounts of the Treasurer and the Business Manager, and certify their audit in a written report to the Society at the final business session. The Council may at its discretion authorize an audit by a certified public account as well as an auditing committee. Such audit shall be published in the official journal.

6. SUBMISSION OF PAPERS AND ABSTRACTS

(a) Three typewritten copies of each abstract of a paper to be presented at the annual meeting must be submitted to the Secretary on or before the closing date specified in the Preliminary Announcement. This date shall be set by the Secretary and when possible shall be sixty days prior to the annual meeting. The Secretary shall return to the authors all abstracts received after the closing date.

(b) At least one author of each paper must be a member of the Society. An author cannot appear on more than two papers.

(c) The abstracts must be in clear, concise English and contain no tabular data. The text must not exceed 1300 typewriter spaces in length. Only unpublished information may be included. Reports of progress, or of disease occurrence, or of routine tests, or of ordinary control measures are not desired unless new and significant developments are clearly indicated.

(d) The abstracts shall be subject to final acceptance and editing by a review committee appointed by the Editor-in-Chief. Accepted abstracts will be published in PHYTOPATHOLOGY at the expense of the Society.

(e) All copies of the abstract must have the following information: 1) title of paper, 2) name, address, affiliation and membership status of each author, 3) sectional preference, 4) time required for presentation (maximum 15 minutes), 5) projection equipment required, including slide size.

(f) Abstracts of papers invited by the Program Committee, or submitted to other societies for inclusion in joint programs will be published in PHYTOPATHOLOGY provided they meet the above requirements, except that the rules regarding unpublished data and membership in the Society will be waived.

7. DIVISIONS

The following provisions shall govern the organization and regulation of Divisions of the Society.

(a) *Name.* Divisions shall use the name of the parent Society with appropriate geographical term; for example, the American Phytopathological Society, Pacific Division.

(b) *Membership.* Divisions shall elect to full membership only members of The American Phytopathological Society, but each Division may elect associate members under such rules as it may adopt.

(c) *Publication.* Summaries of proceedings of the annual meetings of Divisions shall be published in PHYTOPATHOLOGY at the Society's expense. Abstracts of papers presented at Divisional meetings may also be published in PHYTOPATHOLOGY, subject to the rules governing preparation and publication of abstracts as stated in Rule 6, except that no more than two abstracts presented by any one member (as sole or joint author) at the annual meeting of the society and the corresponding annual meeting of the Divisions shall be published at the Society's expense.

(d) *Constitution.* The constitution or articles of organization of all Divisions shall contain a provision or provisions ratifying the above rules. The constitution of all Divisions shall contain nothing in conflict with the constitution of The American Phytopathological Society. With the exceptions defined by the above rules, the Divisions shall enjoy complete autonomy.

8. AFFILIATED SOCIETIES

The following provisions shall govern the establishment of affiliated relations with other phytopathological societies.

(a) Upon advice to the Council that affiliation is desired, the Council may invite to affiliation with The American Phytopathological Society any other professional society in the field of plant pathology.

(b) The existence of affiliation of other societies with this Society shall be indicated by listing the names of affiliated societies in each number of PHYTOPATHOLOGY.

(c) The Council may at its discretion authorize the publication of summaries of the proceedings of annual meetings of affiliated societies in PHYTOPATHOLOGY.

(d) The Council may at its discretion authorize the publication for limited periods, subject to annual renewal of the privilege, of research papers of members of affiliated societies who are not members of The American Phytopathological Society.

9. SOCIETY REPRESENTATIVES IN OTHER ORGANIZATIONS

The Council is authorized to select, with the approval of the Society, representatives in other organizations in which the Society is entitled to representation. The following are specifically authorized: two representatives on the Council of the American Association for the Advancement of Science for a two-year term; one representative on the Division of Biology and Agriculture of the National Research Council for a three-year term; one member of the Editorial Board of the American Journal of Botany for a three-year term; and one representative on the Board of Governors of the American Institute of Biological Sciences for a term of four years.

10. AMENDMENTS

These rules may be amended by a majority vote of the members voting at any regular meeting of the Society, or by a majority vote of the members voting in a ballot conducted by mail.